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LUDWIG'S ANGINA

INTRA-ORAL INCISION IN INFECTIONS OF THE FLOOR OF THE MOUTH

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HISTORICAL DATA

Almost one hundred years ago (1836) Ludwig [1] published his observations on the oral inflammation that later was to bear his name. In the years immediately following, considerable publicity was given to this malady, but it was not until half a century later that the interesting discussions of this type of angina by the French Surgical Society occurred. At this time there was so much debate regarding the propriety of considering the disease an entity that two opposing factions were born. One, headed by Delorme, advocated the idea; the other, sponsored by Nélaton, was opposed. Early in the twentieth century, the publications in America by Davis,[2] Thomas [3] and Price [4] resulted in further discussion and aided greatly in the present-day conception of the disease. As a result, at the present time the term Ludwig's angina has practically universal acceptance.

INCIDENCE OF THE DISEASE; AGE; SEX

Cases that fulfil the requirements for the diagnosis of Ludwig's angina are not common. Over a period of eight years, from 1922 to 1930, the general surgical service at the hospital of the University of Pennsylvania had but twelve cases of this disease in a total of 18,705 patients admitted to this service, or a percentage of 0.07. These figures do not include the patients admitted to the gynecologic, ophthalmologic or otolaryngologic services, which would reduce the percentage still further. Three additional cases were handled by the department of

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Presented as a Candidate's Thesis to the American Laryngological, Rhinological and Otological Society.

Dr. E. L. Eliason, Dr. George P. Müller and Dr. I. S. Ravdin allowed me to use the material from their surgical services at the University Hospital. Dr. E. R. Clark, professor of anatomy, made the anatomic studies possible.

1. Ludwig, K. F. W.: Med. Cor.-Bl. d. Württemb. Arztl. Vereins, Stuttgart **6**:21, 1836.
2. Davis, G. G.: Ann. Surg. **44**:175, 1906.
3. Thomas, T. T.: Ann. Surg. **47**:161, 1908; **48**:788, 1908.
4. Price, J. W.: Ann. Surg. **48**:649, 1908.

otolaryngology and are mentioned in some detail in this article. In toto, the article deals with data secured from fifteen cases that are properly classified as Ludwig's angina (see the accompanying table).

Four of the cases occurred in the first decade, seven between the ages of 10 and 30 years and the remaining five between 30 and 60 years. There was only one female patient in this series. From these figures it would appear that the disease is most frequent in the young and in young adults, and that males are more often attacked. In Ashhurst's [5] series of eighteen cases there were twelve male and six female patients, and seven cases occurred before the age of 30, the remaining eleven appearing between the ages of 30 and 80. By combining the figures of these two series, one could estimate that 55 per cent of all cases occur in the first three decades, although no age seems to be immune.

In the series reported here the duration of the infection until the time of operation varied from two to twenty days, with a seven day average. In all cases the temperature was elevated, the range varying from 100 to 107 F. Leukocytosis was the rule, the counts ranging from 10,000 to 37,500 per cubic millimeter, with an average of 20,500 per cubic millimeter.

Recovery occurred in all but one case. The patient in this case was admitted in a moribund condition and died several hours after admission following an emergency tracheotomy. External operations were performed in eleven cases and internal in two, and in one evacuation was spontaneous. In twelve of the thirteen cases in which operations were performed sublingual pus was found. In one cellulitis only was present. In nine of the fifteen cases there was a definite history of dental caries; two patients gave negative dental histories, and in four cases this rather important fact was not mentioned in the history, so that in but two cases of the series can dental infection be definitely excluded.

DEFINITIONS AND PATHOGENESIS

Various authors have defined Ludwig's angina differently.

Davis [2] stated that it is "a septic inflammation involving the cellular tissues beneath the tongue and jaw, around the submaxillary gland and upper part of the neck."

Ashhurst [5] said that "It is an acute inflammatory process involving the cellular tissues of the floor of the mouth and the submaxillary region on one or both sides of the neck."

Blassingame [6] considered it "a phlegmonous process arising from infections within the floor of the mouth and localized in a definite anatomic space."

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5. Ashhurst, A. P. C.: Ludwig's Angina, Arch. Surg. **18**:2047 (May) 1929.
6. Blassingame, C. D.: Tr. Am. Laryng., Rhin. & Otol. Soc. **34**:33, 1928.

Summary of Cases *

No.	Name	Age	Sex	Chief Complaint	Cause	Operation	Findings	Result	Days in Hospital	Organisms
1	J. B.	22	M	Swelling of floor of mouth	Dental caries	External	Pus and necrotic tissue	Recovery	12	Not stated
2	M. S.	10	M	Swelling of tongue and sublingual tissues	Not stated	External	Not stated	Recovery	13	Not stated
3	C. R.	8	M	Swelling of tongue, sublingual tissues and neck	Not stated	External	Pus and necrotic tissue	Recovery	5	Streptococcus infrequens
4	J. L.	20	M	Lingual and sublingual swelling	Dental caries	External	Abscess, sublingual	Recovery	6	Not stated
5	R. K.	4½	M	Lingual and sublingual swelling	Not stated	External	Sublingual abscess	Recovery	9	Hem. staph. alb.
6	R. G.	43	F	Sublingual swelling	Dental caries	External	Sublingual abscess	Recovery	11	Not stated
7†	W. H.	49	M	Sublingual swelling	Dental caries	External	Pus and necrosis	Recovery	6	Not stated
8	A. S.	1½	M	Cervical adenitis; lingual and sublingual swelling	Not stated	External	Sublingual abscess	Recovery	4	Not stated
9	H. S.	33	M	Cervical and sublingual swelling	Dental caries	External	Cellulitis; no pus	Recovery	6	Not stated
10	M.-T.	57	M	Sublingual and cervical swelling	Dental caries	External	Sublingual pus	Recovery	6	Nonhem. staph. aur.
11	J. F.	11	M	Swelling of neck and tongue	Dental caries	External	Sublingual pus	Recovery	6	Not stated
12‡	H. S.	22	M	Marked dyspnea; swelling of neck and tongue	Admitted in coma	.....	Died several hours after tracheotomy	.....	..	No other operation
13	A. R.	42	M	Sublingual and submaxillary swelling	None found	Internal	Sublingual pus	Recovery	3	No culture
14	R. P.	25	M	Dysphagia; sublingual and submaxillary swelling	Dental caries	None	Spontaneous evacuation of sublingual abscess	Recovery	14	
15	J. M.	13	M	Sublingual swelling	Dental caries	Internal	Sublingual abscess	Recovery	8	Nonhem. strep.

* The ages range from 57 to 1½ years, 73 per cent in the first thirty years. All but one were male patients. Recovery occurred in all cases except the one in which the patient was admitted in coma.

† Follow-up in this case showed that the patient had two subsequent operations in other hospitals for infections of the mouth, and died four months later during an attack.

‡ Autopsy showed "edema of the glottis." There was a necrotic abscess at the base of the tongue on the right side.

Fetterolf [7] has concisely referred to it as "a phlegmon in the floor of the mouth above the mylo-hyoid muscle."

These definitions all agree that the sublingual space must be involved to constitute true Ludwig's angina. Two demand an inflammatory reaction in the upper part of the neck. In all probability all these definitions are correct and also that, in the earliest stages, the disease is confined to the floor of the mouth, the submandibular involvement being a later manifestation of spread by contiguity. The idea of Thomas,[3] of a retrograde infection invading the floor of the mouth from the superficial part of the submaxillary salivary gland, has not been confirmed by the findings of various surgeons or by the injection experiments of Blassingame.[6]

Blassingame found that in dogs: 1. Ink injected under the mucosa of the posterior part of the mouth passed through the floor of the mouth to the deep cervical lymph nodes. 2. Material injected into the peritonsillar region did not enter the floor of the mouth, but passed to the deep cervical nodes. 3. Ink injected into the anterior floor of the mouth and into the mylohyoid in this region did not pass backward, but ran into the lymphatics superficial to the mylohyoid muscle, eventually reaching the small lymph nodes adjacent to the superficial part of the submaxillary salivary gland. He concluded from these experiments that infection from the posterior teeth or that part of the jaw may: (1) extend into the sublingual space and cause Ludwig's angina; (2) pass through this space to the deep cervical nodes and (3) involve both these areas. The tonsils and anterior teeth are not likely to be causative factors.

Ashhurst [5] emphasized the fact that this condition is a cellulitis and not a lymphadenitis. In eleven of eighteen cases reported by him a definite history of dental caries was associated, and of the seven remaining cases, in only three could dental caries be absolutely excluded. It should be remembered that lymphadenitis per se does not cause sublingual or submaxillary cellulitis unless a gland has suppurated, broken through its capsule and caused a local cellulitis by direct extension to the surrounding cellular structures. In order to have cellulitis one must have a local infection, which is then transmitted to the cellular tissues by contiguity and not through lymphatic channels. In eight of the fifteen cases here reported, a history of dental caries was present. In only two of the remaining seven could it be definitely excluded.

In Ashhurst's injections, performed on a cadaver, from 40 to 45 cc. of dilute chinese ink was injected under the mucosa of the floor of the mouth. The result was that the tongue was pushed against the palate, and almost at once the dye appeared in the submaxillary region beneath the deep fascia. The undersurface of the mylohyoid remained

7. Fetterolf, George: Tr. Coll. Phys., Philadelphia **51**:312, 1929.

unstained except at the posterior border. The submaxillary salivary gland itself was not stained, only the fascia between its lobules.

ANATOMY

To understand the course of the disease, an understanding of the anatomy is essential. It will be recalled that the sublingual space is bounded laterally and in front by the body of the mandible. Below is found a firm diaphragm made up of the two mylohyoid muscles fused in a median raphe, which are attached laterally and in front to the inner surface of the mandible and posteriorly to the hyoid bone. These muscles are enclosed in a definite and fairly dense sheath, which, near their bony attachment, thins out in a firm unyielding aponeurosis. External to this muscle is the deep cervical fascia, which, although rather thin, is strong and unyielding. The fascia is attached to the inferior edge of the mandible and splits to envelop the anterior bellies of the digastric muscles, all of which give additional firmness to the floor of the mouth. In addition, an inward partition from the fascia completes the capsule of the submaxillary salivary gland and separates it from the parotid. Still further externally are found the platysma muscle, superficial fascia and skin.

From the posterior borders of the mylohyoids are continued the pharyngeal constrictors, separating the pharynx from the neck. A deficiency exists between the middle constrictors and the mylohyoids, giving rise to an opening extending laterally from the hyoid bone to the inner side of the mandible near the angle. This gap is principally filled by the hyoglossus muscle and the curve of the horseshoe-shaped submaxillary salivary gland, which unites the superficial and deep portions of that structure. Additional, less bulky structures passing through this space are the glossopharyngeal and hypoglossal nerves, the lingual artery and vein and the styloglossus muscle.

The posterior boundaries of the sublingual space are less easily visualized, but are just as definite. As the tongue dips downward and backward toward the epiglottis, the free edges found further forward in its more motile portion no longer exist. The palatoglossus and the more robust styloglossus have attached themselves at each side and constitute a firm, mucous membrane-covered, muscular ridge.

The superior boundaries of this space are simply the tongue and mucosa of the floor of the mouth.

Taking into consideration these details of anatomy, it does not seem strange that Ludwig's angina should act as it does. Inflammation of this region should cause a boardlike hardness under the mandible on account of the tenseness of the mylohyoid muscles and the deep cervical fascia. It should cause swelling of the floor of the mouth and push the

tongue up and back. When one realizes that the distance from the symphysis of the jaw bone to the base of the epiglottis in the adult is but 2½ inches (6.27 cm.), it is not remarkable that edema of the larynx should follow an unrelieved condition of this sort. Nor is it uncanny that pus should often be found in the neck in the region of the superficial part of the submaxillary salivary gland when a definite anatomic communication exists between this region and the site of initial infection.

It is remarkable, however, that more attention has not been given to the relief of this condition by intra-oral operation early in its onset, before much cervical involvement has occurred. In the large series of cases reported by Thomas,[3] there were seventeen cases of spontaneous evacuation. All of the abscesses opened internally; in one there was both an internal and an external opening. Therefore, at least it seems that when spontaneous opening is at all possible nature chooses the internal route. Certain surgeons have made sporadic attempts at intra-oral evacuation of sublingual pus, but the successful cases have received little publicity, and the unsuccessful attempts appear to have been not sufficiently radical.

Nélaton [8] made a sublingual puncture, but found only blood.

Chauvel [9] made a double sublingual puncture and exposed a gangrenous focus above the mylohyoid muscle extending to the upper border of the thyroid cartilage.

DuBois [10] found pus by sublingual incision.

Cuffe [11] found only blood, but later repeated the incision and found pus.

Holthouse [12] scarified only and found no pus.

Ross,[13] by sublingual puncture, found no pus, but later located a large abscess by external operation.

Ripault [14] found pus by both the external and the internal route.

One explanation of the relatively few attempts to evacuate such abscesses by the oral route may be that many of the patients go to the general surgeon, who often has little practice in surgical manipulations inside the mouth and in the use of head mirrors for illumination. The argument that one should hesitate to allow pus to escape into the mouth is scarcely tenable, for no one hesitates to open an abscess in quinsy or a retropharyngeal abscess, and dire results from the pus so evacuated are

8. Nélaton, C.: Bull. et mém. Soc. d. chir. de Paris **18**:489, 1892.
9. Chauvel, J.: Bull. et mém. Soc. d. chir. de Paris **18**:474, 1892.
10. DuBois, M.: Arch. gén. de méd. Paris **13**:81, 1827.
11. Cuffe: Brit. M. J. **2**:733, 1867.
12. Holthouse, C.: Tr. Clin. Soc., 1869, p. 140.
13. Ross, G. G.: Ann. Surg. **33**:720, 1901.
14. Ripault, H.: Ann. d. mal. de l'oreille, du larynx **31**:235, 1895.

quite unusual. If the mouth cannot be opened, as is quite often the case late in the disease, or if pus in the neck is already present, then external opening only will suffice. But if the patient is seen early, there is a possibility of bringing the infection to a speedy termination with relatively simple intra-oral surgical maneuvers. If this fails, no harm has been done, and the external operation can always be performed.

REPORT OF CASES

This method was suggested to me by Dr. George Fetterolf, whom I called in consultation in case 1.

CASE 1.—A young man had a right-sided sublingual swelling, an elevation of temperature, leukocytosis and dysphagia associated with a boardlike hardness of the tissues under the chin and right mandible. The condition was not fulminant, and while the patient was under observation spontaneous perforation of a sublingual abscess occurred through an opening near the right side of the base of the tongue. We resolved at this time to attempt intra-oral drainage in the next case.

CASE 2.—A man, aged 42, came to my office some months after the patient in case 1, complaining of a tickling sensation of the right side of the throat which had been present for four days. There was little to be found on examination, except a slightly swollen right submaxillary gland. He was allowed to go home and was instructed to use hot gargles and heat externally. Two days later I was called to his home and found that marked progression of the condition had occurred. Decided sublingual swelling was present on the right side; the tongue was pushed toward the roof of the mouth and to the left, and marked dysphagia and slight dyspnea were symptoms. The temperature was 101 F., and a boardlike hardness was present under the chin and the right side of the lower jaw. With both the right and the left forefingers inside the mouth, palpation of the floor revealed a decided swelling on the right side. One could not be sure of fluctuation.

The patient was immediately admitted to the hospital of the University of Pennsylvania, where I again called Dr. Fetterolf in consultation. We decided to explore the sublingual region through the mouth. At my request, Dr. Fetterolf performed the operation and incised the mucosa over the point of greatest swelling, which was about the level of the second molar tooth and half way between the frenum and the alveolar ridge. At a very slight distance beneath the mucosa the scalpel entered an abscess cavity, and pus began to escape. A hemostat was then introduced, the opening dilated and several tablespoonfuls of pus evacuated. There was a rapid subsidence of symptoms after the evacuation, and the patient was allowed to leave the hospital on the third day.

CASE 3.—A boy, aged 13 years, was admitted to the University Hospital in January, 1930, with a history of sore throat and dysphagia. The onset of these symptoms had occurred six days before admission and they had become progressively worse. Two days before admission swelling of the tongue and a lump under the jaw appeared.

On admission it was noted that the tongue was swollen, associated with slight restriction of movement and swelling of the sublingual tissues. Below the chin the tissues were hard and swollen, and the right submaxillary salivary gland and lymph glands of that region were swollen and tender.

The day after admission there seemed to be some improvement; swallowing was performed with greater ease, and the general prostration was not as great. The next day, however, the patient became rapidly worse. His temperature, which previously had not been above 100 F., jumped to 102 F., with a proportionate acceleration of the pulse rate. The swelling increased rapidly, and slight respiratory embarrassment was present. The leukocytes numbered 24,000. The following note was entered by me on his history:

"Palpation of the sublingual tissues and floor of the mouth today showed evidence of swelling as the finger was passed from before backward. This swelling was more marked on the right side. Swelling below the chin was more marked today." On account of the increase of swelling, elevation of the tongue and marked dysphagia, drainage was considered imperative. An incision was made through the mucous membrane on the right side about 1½ inches (3.77 cm.) behind the lower anterior teeth and one-fourth inch (0.64 cm.) from the midline. This incision was little more than a puncture. Through this opening a small hemostat was inserted and gently pushed toward the base of the tongue as well as toward the midline. After the penetration of about one-fourth inch of tissue, the sensation of the forceps entering a cavity was felt. The points were spread, and about 2 ounces (59 cc.) of foul, thick pus escaped.

During the next twelve hours the tongue and the submaxillary and sublingual regions became still more swollen. A Kelly hemostat was introduced into the wound, the points spread and more pus evacuated. A small rubber dam was inserted. From this time improvement was so rapid and uninterrupted that the patient was discharged with almost complete absence of swelling the sixth day after operation.

These three cases fulfil the requirements of Ludwig's angina if it is admitted that the condition can exist with sublingual pus alone. In case pus in both the sublingual space and the neck is insisted on, preceded by sublingual infection, it would probably be proper to consider these cases as early stages of Ludwig's angina. If the pus must form around the submaxillary salivary gland and reach the sublingual space by retrogression, then they are not properly included in the Ludwig category. Regardless of the nomenclature and classification, if the foregoing symptom complex is present, experience in these few cases seems to indicate that the sublingual method of drainage is worth a trial. If cellulitis alone is present, without abscess formation, failure will probably result, but no harm has been done, and one of the effective external operations can still be performed. One signal advantage of this operation is its simplicity, with the result that it will often be called on more quickly than the more formidable external operation, which often is properly delayed in the hope that spontaneous subsidence will occur. No anesthesia is required, and the associated pain is probably not as great as that experienced in the opening of an abscess in quinsy. If the condition is so far advanced that the mouth cannot be opened, the external drainage route is the only one possible.

CONCLUSIONS

1. Fifteen cases of Ludwig's angina are reported, with recovery in fourteen.
2. The series indicates that the young adult male is most often affected.
3. External operations were performed in eleven cases, with recovery in all.
4. One patient recovered by spontaneous internal evacuation.
5. Two patients recovered promptly following intra-oral drainage.
6. Dental caries was found to be the usual etiologic factor.

ILLUSTRATIVE SECTIONS THROUGH THE SUBLINGUAL SPACES

The following figures are photographs of frontal serial sections of a formaldehydized frozen head, showing the region mentioned in the text.

Fig. 1.—Through the symphysis of the mandible and the anterior limit of the sublingual space. In this and the following pictures, *T* indicates the tongue; *M*, mandible; *GG*, genioglossus muscle; *SM*, sublingual mucosa; *D*, digastric muscle; *SG*, sublingual salivary gland; *GH*, geniohyoid muscle; *MH*, mylohyoid muscle; *SMG*, submaxillary salivary gland; *CF*, cervical fascia; *PG*, palatoglossus muscle; *SMG 1*, deep portion of submaxillary salivary gland; *SMG 2*, superficial portion of submaxillary salivary gland; *H*, hyoid bone.

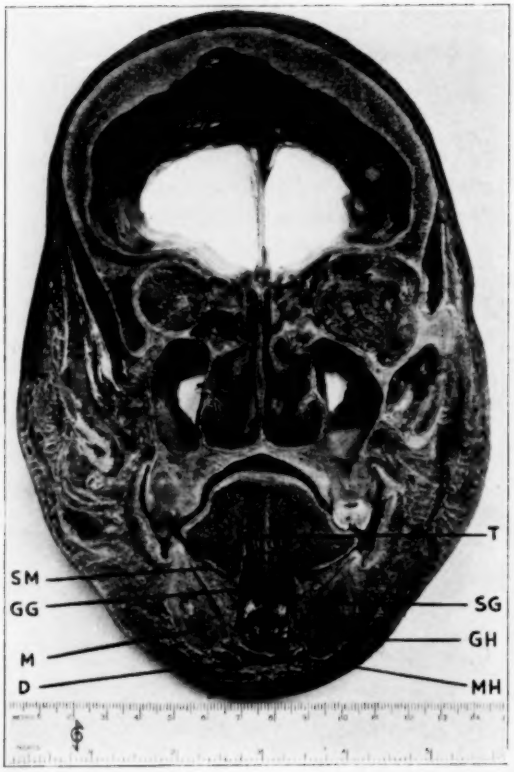

Fig. 2.—The sublingual gland here occupies most of the sublingual space. The aponeurosis of the mylohyoid can be seen extending from one jaw bone to the other, with the geniohyoid and genioglossus muscles above and the bellies of the diagastric below.

Fig. 3.—There is little difference between this region and that pictured in figure 2, except that the mylohyoid muscle appears to be more robust.

Fig. 4.—The mylohyoid muscle is here decidedly increased in size. The glands shown are the deep portion of the submaxillary salivary glands.

Fig. 5.—One-half inch posterior to the section in figure 4; the same structures are seen as before, the only difference being that of size. It is in this region that intra-oral incision was made in my cases.

Fig. 6.—Still more posterior, the submaxillary gland is much smaller. The palatoglossus fibers, having entered the tongue, form an effective posterolateral barrier to the sublingual space. The superficial portion of the submaxillary gland can be seen on the left side of the photograph.

Fig. 7.—On the right side of the picture the saw passed through the extreme posterior portion of the mylohyoid. Here can be seen the junction of the deep and superficial portions of the submaxillary salivary gland.

SPONTANEOUS PERFORATION OF BENIGN STRICTURE OF THE ESOPHAGUS

REPORT OF A CASE

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The ingestion of lye is the most common cause of benign stricture of the esophagus, and a small amount of a dilute solution of this substance will usually produce marked scarring in that organ.

When the solution is concentrated and has been taken with suicidal intent, the entire esophagus may be practically destroyed with resulting mediastinitis and death, and weaker solutions taken in smaller amounts may produce sufficient ulceration of the esophagus to be followed by the formation of a periesophageal abscess. If the inflammatory process has extended beyond the wall of the esophagus, the initial passage of dilating sounds may rupture the wall of the abscess and produce general mediastinitis with fatal termination. Fortunately, this complication rarely arises, and it has never been my experience to perforate a benign esophageal stricture resulting from lye after the first dilatation. It is not difficult to understand why perforation rarely occurs when one examines the density of scars produced in the esophagus by the ingestion of a caustic solution. Spontaneous perforation of a normal esophagus may occur as the result of prolonged vomiting. The ingestion of a foreign body such as a sharp piece of bone may perforate a normal esophagus or one that has been scarred from various irritants and infections, but spontaneous rupture of a benign stricture of the esophagus from swallowing ordinary food is observed very rarely. The case reported herewith is illustrative of this complication.

REPORT OF CASE

At the age of 18 months, the patient had swallowed less than a teaspoonful of a solution of lye. For a month following the burn she was able to swallow soft food, but after dilatation of the stricture was attempted, obstruction became complete and gastrostomy was performed. During the succeeding four years numerous attempts had been made to restore the lumen of the esophagus, and the esophagoscope was passed more than a hundred times without benefit.

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In April, 1918, the patient was brought to the Mayo Clinic for treatment, and after a thread had been swallowed, the strictures were dilated and the gastrostomy opening was closed. The scars in the esophagus were very dense, and it required considerable pressure to force dilating sounds through the stricture. Repeated dilatations were carried out for thirteen years, and although the patient was quite normal, it was necessary to continue to pass sounds every two or three months to permit free deglutition. Many times during this period a bolus of food would become obstructed at one of the strictures, and increased efforts at swallowing were required to force it into the stomach.

The last time the stricture was dilated was on Sept. 11, 1931. On November 11, the patient was eating luncheon rather rapidly and a piece of meat became impacted in the esophagus. Ordinary efforts at swallowing failed to remove it during the afternoon and evening. The following morning the obstruction was still present, and the patient drank water and employed unusual force in swallowing in an attempt to dislodge the food from the stricture. This effort was immediately

Diffuse scarring of the esophagus secondary to the ingestion of lye with recent perforation.

followed by severe pain in the back, and when I examined her an hour later she was in a state of shock. Esophagoscopic examination was made at once, and although a small amount of blood was coming from the lumen of a stricture in the middle of the esophagus, a foreign body could not be seen. The patient became progressively worse, with signs of pneumothorax on the left associated with effusion, and died on November 13.

At necropsy, a perforation was found in the upper portion of a dense scar in the esophagus which had been followed by mediastinal and left pyopneumothorax (see illustration).

COMMENT

It is likely that impaction of food in the lumen of the stricture had reduced the blood supply to that area to such a degree that necrosis had developed, resulting in weakening of the wall of the esophagus so that perforation occurred when unusual efforts at swallowing were employed.

LARYNGEAL TUBERCULOSIS

SOME EXPERIENCES AND OBSERVATIONS

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The material that forms the basis for this study was seen at the Kings County Hospital and the Metropolitan Hospital in New York on the service of Dr. Mervin C. Myerson. In these general hospitals, which are maintained as public institutions, a majority of the patients are from the poorer strata of society. They comprise laborers, domestics, etc., including many illiterates and persons of the immigrant classes. It is therefore readily understood that many come there as neglected patients and in the late stage of their disease. This paper represents my experiences with such cases, in addition to a review of the literature, past and present.

Historically speaking, a condition that may have been laryngeal tuberculosis was mentioned by Hippocrates.[1] Petit [2] (1790) and Sauvée [3] gave a comprehensive description of this condition in their writings, but did not differentiate their cases from those of syphilis. In 1825 the researches of Louis [4] brought out a factor that is accepted today: that the mechanical irritation of pulmonary secretions causes the laryngeal infection. Albers [5] first attempted to differentiate syphilis from tuberculosis of the larynx, and advocated tracheotomy to afford complete rest.

Thesis submitted to the Faculty of Otolaryngology of the Graduate School of Medicine, the University of Pennsylvania, in partial fulfilment of the requirements for the degree of Master of Medical Science (M.Sc. [Med.]) for graduate work in otolaryngology. The degree was granted Feb. 20, 1932.

Involvement of the larynx is one of the most serious complications of pulmonary tuberculosis. The writer was prompted to undertake a study of this subject because it was felt that laryngologists and pulmonary specialists do not give this laryngeal disease sufficient consideration.

1. Quoted by Wright, Jonathan: A History of Laryngology and Rhinology, ed. 2, Philadelphia, Lea & Febiger, 1914, p. 193.

2. Petit, M. A.: De phthisis laryngea, Montpelier, J. F. Tournel, 1790.

3. Sauvée, A.: Recherches sur la phthisie laryngée, Paris, an X, 1802, vol. 9.

4. Louis, P. C. A.: Recherches anatomico-pathologique sur la phthisis, Paris, 1825; translated by Charles Cowan, revised by H. Bowditch, Boston, Hilliard Gray & Co., 1836, p. 47.

5. Albers, J. F. H.: Die Pathologie und Therapie der Kehlkopfkrankheiten, Leipzig, C. Cenobloch, 1829, p. 176.

In a prize essay written in 1837, Trousseau and Belloc [6] stated that laryngeal tuberculosis usually followed pulmonary tuberculosis, but that occasionally the primary lesion was localized in the larynx. They were the first exponents of rational therapy for diseases of the larynx; they advocated sprays, swabbing and auto-insufflation of powders through curved cannulas.

Rokitansky [7] (1846) began the study of morbid pathology and looked on the tubercle as "coagulated protein stuff" which broke down and formed ulcerations. Rheiner,[8] seven years later, "advanced the theory that tuberculous ulcerations of the larynx were dependent on mechanical injury because of the anatomic relationship of the parts, especially when these favored areas had been previously reduced in resistance through injury or inflammation."

It is interesting at this point to note what early efforts were made to study the laryngeal structures. Bozzini [1] (1807), a vocal teacher, became interested in seeing what happened in the larynx while singing. He was the first to devise the laryngoscope. It consisted of a mirror with a wax candle and a reflector. Manuel Garcia, the father of laryngology, contributed the first practical laryngoscope in 1855.[9] In his thesis, Gerhardt [10] reported a large series of cases in which Garcia's method of examination was used, and he clearly described syphilis and succeeded in separating it from phthisis.

Virchow [11] (1864-65) vigorously opposed Louis' opinion regarding the mechanical cause of phthisical ulcers; he claimed that "laryngeal tuberculosis was due to the tubercle and nothing else," and reasserted the fact that ulceration is consequent on destruction of the tubercle. In 1879, Oscar Heinze [12] demonstrated that the cause of laryngeal tuberculosis is tuberculous infiltration of the mucosa. As early as 1846,

6. Trousseau, A., and Belloc, H.: A Practical Treatise on Laryngeal Phthisis, Chronic Laryngitis and Diseases of the Voice, translated by J. A. Warder, Philadelphia, A. Waldie, 1839, p. 13.

7. Rokitansky, K.: Manual of Pathological Anatomy, London, Sydenham Society, 1852, vol. 4, p. 32.

8. Rheiner, C.: Ueber der Ulcerations Process im Larynx, Virchows Arch. f. path. Anat. **5**:534, 1853.

9. Newcomb, James: Manuel Garcia, Teacher, Discoverer and Man, New York, William Wood & Company, 1905, p. 1; M. Rec. **67**:441 (March 25) 1905.

10. Gerhardt, C., and Roth: Ueber syphilitische Krankheiten des Kehlkopfes, Virchows Arch. f. path. Anat. **21**:7, 1861.

11. Virchow, R.: Handbuch der speziellen Pathologie und Therapie, Erlangen, Ferdinand Enke, 1854, p. 461.

12. Heinze, O.: Der Kehlkopfsschwindsuch nach Untersuchungen im Pathologischen Institute der Universität Leipzig, Leipzig, Veit & Company, 1879, p. 76.

Horace Green [13] introduced applications of silver nitrate to the areas involved in this disease. This was met with considerable opposition and criticism. William Marcet,[14] twenty-three years later, in 1869, advocated scarification and incision. Curettage was next introduced by Heryng,[15] and advocated by Herman Krause [16] in 1885.

ETIOLOGY

It was conclusively shown by Virchow [11] (1865) and Heinze [12] (1879) that laryngeal tuberculosis was due to the tubercle bacillus, but the problem of the route of the invasion of the larynx was of great concern in the early years of the study of this infection.

There have always been two theories: one that the primary or initial lesion may occur in the nose, pharynx or larynx; the other that the primary lesion always originates in the lung and that infection of the larynx and upper respiratory tract is secondary and due to direct contact with infected sputum or by way of the lymph or blood channels.

George B. Wood [17] believes that primary tuberculosis of the larynx has unquestionably occurred, but that it is extremely rare. Lockard [18] stated that "until a case of laryngeal phthisis, unaccompanied by either pulmonary or lymphatic disease has been proved, the assumption of primary infection cannot be maintained." Thomson [19] said: "Primary tuberculosis of the larynx as shown by postmortem integrity of the lungs is so extremely rare that for practical purposes its possibility may be neglected."

Arrowsmith [20] stated: "It is conceded today that primary laryngeal tuberculosis probably never occurred and that most individuals who exhibit tuberculous manifestations in mature life have been infected in childhood or infancy, the infection remaining latent as a rule, until some

13. Green, Horace: Treatise on Diseases of the Air Passages, Comprising an Inquiry into the History, Pathology, Causes and Treatment of Those Affections of the Throat Called Bronchitis, Chronic Laryngitis, Clergyman's Sore Throat, etc., New York, Wiley and Putnam, 1846, p. 201.

14. Marcet, William: Clinical Notes on Diseases of the Larynx, London, Longmans, 1869.

15. Heryng, T.: Behandlung der tuberkulöse Larynxphthise, Deutsche med. Wchnschr. **13**:126, 1887.

16. Krause, Herman: Berl. klin. Wchnschr. **22**:462, 1885.

17. Wood, George B.: The Etiology of Tuberculosis in Upper Respiratory Tract, Tr. Am. Laryng., Rhin. & Otol. Soc., 1907, p. 264.

18. Lockard, L. B.: Tuberculosis of the Nose and Throat, St. Louis, C. V. Mosby Company, 1909, p. 46.

19. Thomson, St. Clair: Diseases of the Nose and Throat, ed. 2, New York, D. Appleton and Company, 1916.

20. Arrowsmith, H.: Present Day Aspects of Laryngeal Tuberculosis, Tr. Am. Laryng. A. **35**:282, 1917.

adventitious stimulus spurs it into activity in any or all of its protean forms." Spencer [21] concluded, "with an experience of more than 20 years, I have never seen a primary case."

With the present-day acceptance of laryngeal tuberculosis as a secondary lesion, opinions as to the route of infection are varied. Ballenger,[22] Bonney [23] and Dworetsky [24] believe that owing to the anatomic structure of the larynx and its position in the respiratory tract, bacteria-laden sputum passing by and resting in the laryngeal mucosa causes the infection. Supporting this theory are the following facts:

1. Localized lesions occur in the larynx most frequently at points that are most subject to the irritation of passing secretion.

2. Tubercle bacilli are known to penetrate normal mucosa without leaving any trace of their entrance.

3. Tuberculous laryngitis rarely occurs in infants and children who are suffering with tuberculous cervical adenitis.

Heinze,[12] Krieg [25] and Coakley [26] contend that the route of infection is most often through the lymphatics. Various statistics are quoted to support this theory: 1. Magenau [27] claimed that 40 per cent of unilateral lesions of the larynx occur on the same side as the pulmonary changes or the most changes, while Krieg [25] found 90 per cent. 2. The tubercles show a subepithelial distribution, decreasing in number as they approach the mucosa; none occur in the epithelial layer.

Most clinicians and anatomists look on the sputal infection as the predominant route.

Sex.—Thomson [28] found laryngeal tuberculosis about evenly divided in males and females; Dworetsky [29] found a small percentage more in males; Spencer [21] noted 3 males to each 2 females. There were 3 males to every 2 females in my cases.

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21. Spencer, F. R.: Laryngeal Tuberculosis, Philadelphia, J. B. Lippincott Company, 1927, p. 4.

22. Ballenger, W. L.: Diseases of the Nose and Throat, ed. 6, Philadelphia, Lea & Febiger, 1930, p. 727.

23. Bonney, S. G.: Pulmonary Tuberculosis and Its Complications, ed. 2, Philadelphia, W. B. Saunders Company, 1910, p. 642.

24. Dworetsky, J. P.: Etiology and Prophylaxis of Tuberculous Laryngitis, Ann. Otol., Rhin. & Laryng. **23**:835, 1914.

25. Krieg: Arch. f. Laryng. u. Rhin. **8**:519, 1898.

26. Coakley, C. G.: Diseases of the Nose and Throat, ed. 7, Philadelphia, Lea & Febiger, 1930, p. 533.

27. Magenau: Arch. f. Laryng. u. Rhin. **9**:304, 1899.

28. Thomson, St. Clair: Tuberculosis of the Larynx: Ten Years' Experience in a Sanatorium, London, His Majesty's Stationary Office, 1924, p. 9.

29. Dworetsky, J. P.: How to Prevent Tuberculosis of the Larynx, J. Outdoor Life, October, 1915, p. 2.

Age.—The majority of cases occur between the ages of 20 and 45. Spencer [21] stated that the condition occurs "rather frequently in patients past 45," and Thomson [30] reported a case in a patient at the age of 63. In this series there was a man aged 74. Dworetzky [24] did not find any cases among 100 children in his series of 600 patients with pulmonary tuberculosis.

Predisposing Causes.—In the past few years much attention has been given to the existence of nasal and pharyngeal infection as a predisposing cause of laryngeal tuberculosis. It has been claimed that an infection of the sinuses or throat often causes a catarrhal condition of the larynx and thus lowers the resistance of the laryngeal mucosa.

Thomson [28] concluded, after five years of careful study of 1,456 tuberculous patients, that "the affections of the nose and pharynx are no more frequent in patients with tuberculosis of the larynx than in those where the disease is limited to the lung."

On the other hand, Dworetsky [29] found "93 per cent of patients with tuberculous laryngitis had a greater or less obstruction or disease in the nasal passages and hence an interference with normal breathing, while 80 per cent showed marked pharyngitis with post nasal catarrh and 60 per cent gave a history of susceptibility to colds which causes repeated inflammatory attacks in the nose and throat."

Of the cases in this study, a majority showed some degree of disease of the nose and throat.

Occupation.—Lockard's [18] table giving the occupations of 1,280 patients with laryngeal tuberculosis shows a corresponding relationship with the occupations of those afflicted with pulmonary tuberculosis. Laryngeal tuberculosis was more frequently seen in unhealthy indoor surroundings, although a large number of cases occurred in persons engaged in outdoor occupations. A relatively small number occurred among actors, singers and preachers.

It must be remembered that the occupations reported depend on the type of institution in which the patients were seen.

PATHOLOGY

The pathologic changes of tuberculosis assume the same protean forms in the larynx as in other organs. One must bear in mind the anatomy and physiology of the larynx that is altered by the infection. Histologically, the lesion is an inflammatory response to an irritant toxin. A spherical area is formed of whirling layers of modified cells

30. Thomson, St. Clair: Tuberculosis of the Larynx in a Gentleman Aged Sixty-Three, Under Treatment with Galvanocautery, Proc. Roy. Soc. Med. (Sect. Laryng.) **11**:15, 1917.

resembling epithelial cells in their early state and therefore designated "epithelioid cells." These have invested and engulfed one or more tubercle bacilli. Frequently near the center there are one or more multinuclear giant cells of Langhans in which tubercle bacilli are oftentimes found. About the periphery there is a zone of small round lymphocytes, which are derived from the nearby blood vessels. Because the tubercle is avascular, coagulation necrosis occurs in its center. The characteristic location of the lesion is in the subepithelial layer. The epithelium is not involved unless it is attacked by the underlying disease process. In such a case the superficial epithelium is lost and an ulcer results.

Types of Lesions.—Hyperemia or anemia may be manifest, but cannot be called true evidence of laryngeal tuberculosis. The typical lesion begins as a thickening and swelling of the submucosa which are caused by an infiltration of round cells and tubercles. The epithelial covering is usually thickened, and there may be some degree of edema in the subepithelial tissue.

The clinical picture varies according to the amount of involvement and the location of the lesion. Where there is a greater amount of loose areolar tissue, as in the aryepiglottic folds, interarytenoid sulcus and arytenoid area, there is usually more swelling. There is less in the regions of the cords, the ventricular bands and the lingual surface of the epiglottis where the submucosa is thinner.

Manassa's work on the pathology of laryngeal tuberculosis is classic. Wood [31] stated: "According to Manassa, the edema which is so frequently associated with laryngeal tuberculosis may show histologically either large spaces lined with endothelial cells, which are probably dilated lymph spaces, or another type in which the bundles of connective tissue have been separated by the fluid. Tubercles are usually not found in the immediate neighborhood of edematous tissue."

Ulceration occurs when one or more subepithelial tubercles caseate, breaking through the surface. According to Wood,[31] "Manassa has demonstrated the presence of small ulcers without caseation. The epithelium is apparently destroyed either by pressure or by infiltration of the tuberculous tissue immediately beneath it. Tuberculoma occurs in two forms: one the fibro-tuberculoma with a preponderating connective-tissue element, and the other the granulotuberculoma in which the tubercles are surrounded by granulation tissue." These tuberculomas are occasionally mistaken for malignant growths. When fibrosis occurs, it is a manifestation of healing. In advanced lesions perichondritis and chondritis are seen together. At times there may be necrotic sloughs.

31. Wood, G. B.: Laryngeal Tuberculosis, in Jackson, C., and Coates, G. M.: The Nose, Throat and Ear, and Their Diseases, Philadelphia, W. B. Saunders Company, 1929, p. 868.

Fetterolf [32] classified lesions of tuberculous laryngitis as follows: (1) infiltration with superficial ulceration; (2) infiltration with deep ulceration, and (3) infiltration with superficial and deep ulceration. The following is the classification of E. Kaufman: [33] (1) the lenticular type, which later ulcerates superficially; (2) the infiltrative type, which gives rise to deep ulcers (these are found especially where lymphoid tissue abounds); (3) ulcers with hard borders, the chronic type found in the cords and arytenoids, and (4) the polypoid form, which resembles new growths.

Dworetsky [34] gives the following clinical classification:

1. Peracute tuberculosis of the larynx
2. Acute tuberculosis of the larynx (stage 1, stage 2, stage 3)
3. Subacute tuberculosis of the larynx (stage 1, stage 2, stage 3)
4. Chronic tuberculosis of the larynx (stage 1, stage 2, stage 3)

The regions of the larynx which are most frequently affected are those which are subject to the irritation of passing secretions, namely, the posterior portion of the cords, the interarytenoid area and the arytenoids. The following tabulation demonstrates the frequency and location of lesions in the cases studied:

	No. Times
Cords	38
Interarytenoid space	30
Arytenoids	33
Ventricular band	8
Aryepiglottic fold	8
Epiglottis	20
Subglottic area	2
Tonsil	3
Soft palate	3
Gums	1
Tongue	1

Of these patients only 4 had solitary lesions. The foregoing figures comply in percentage with statistics complied by Gaul, MacKenzie, Lake, Lockard [18] and the Phipps Institute, with the exception of a slightly greater increase in epiglottic involvement.

The stage of the laryngeal condition in the cases studied was: acute tuberculosis of the larynx, 8 cases; subacute tuberculosis, 13 cases, and chronic tuberculosis, 24 cases.

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32. Fetterolf, George E.: The Larynx in One Hundred Cases Dying of Pulmonary Tuberculosis, Tr. Am. Laryng. A., 1914, p. 258.

33. Kaufman, E., quoted by Spencer (footnote 21).

34. Dworetsky, J. P.: Laryngeal Tuberculosis, a Three Years' Study, Dept. of Health, City of New York, Tuberculosis Monograph no. 2, 1916, p. 43.

SYMPTOMATOLOGY

The earliest symptoms may be a tickling in the throat or the sensation of a lump when swallowing. Later an occasional hoarseness, vocal fatigue or slight pain during deglutition may be noted. Solenberger [35] stated: "Sometimes nearly all these external signs are absent in the process of involvement even to the point of disintegration."

Hoarseness is a very common symptom. This occurs when the lesion affects the approximation of the cords. Later dyspnea can occur because of a narrowing of the glottis by infiltrations, edema or granuloma. In one case of this series it was necessary to perform a tracheotomy for the relief of dyspnea. This was due to the presence of a tuberculoma filling most of the glottis.

Odynophagia is a most distressing symptom. It is caused by either contact of food or saliva and mucus with an ulcerated exposed area or contact of an ulcerated area with surrounding tissues. The tissues making contact with the ulcerated area may or may not be ulcerated.

The structures in which ulceration could cause contact with food, mucus and saliva would be: (1) the free border of the epiglottis, (2) the external aspect of the aryepiglottic fold, (3) the external and posterior aspects of the arytenoid and (4) the postcricoid region.

Ulcerated areas that would cause pain by contact in the other tissues of the larynx may be located anywhere within the internal aspect of the larynx. Such areas would be the laryngeal aspect of the epiglottis, the laryngeal aspect of the aryepiglottic fold, the false and true cords and the anterior or mesial aspect of the arytenoid.

Pain robs the patient of his best medicine, food, and if it is not relieved, a vicious cycle is established which rapidly carries him downhill.

The larynx may appear pale. This pallor is to be attributed to the general anemia rather than to the local picture. The larynx is often seen to be inflamed and reddened. This is most frequently due to the excessive cough and the irritating sputum. The earliest pathologic process is most frequently seen to begin as an infiltration of the interarytenoid sulcus. There usually is a pale thickening, and at times a papilliform growth. Infiltration of the arytenoids with edema makes them appear globular or pear-shaped; sometimes this is spoken of as "dumb-bell shaped." The vocal cords thicken and lose their pearly white luster, minute vessels cover their surfaces and their edges may present a "mouse-nibbled" appearance, or a punched-out section may be seen which is an ulcer.

35. Solenberger, A. R.: Prophylaxis of Laryngeal Tuberculosis, Tr. Am. Laryng., Rhin. & Otol. Soc. **18**:480, 1912.

Tuberculomas may occur in any portion of the larynx; these irregular granulation-like masses are often a late manifestation, most frequently interarytenoid and subglottic.

Edema produces a pale grayish color of the mucosa.

Early ulcerations are usually superficial. The deep ulcers are a later manifestation and are more frequent where the submucosa is thicker. The edges are irregular; the base is covered with a thin coating of purulent exudate which, when cleaned or wiped off, reveals a granular surface.

Infiltration of the epiglottis begins as a thickening near the rim, usually on one side, and gradually involves the whole free surface. The most frequent site of ulceration is along the rim.

DIAGNOSIS

Greene [36] stated: "Suffice to say, that an early diagnosis of laryngeal tuberculosis ranks second in importance to the early diagnosis of the pulmonary lesion."

Most often the laryngeal picture is so typical that a diagnosis may be made with little difficulty. Spencer [21] believes that in many cases there is "an ashen-gray pallor of the larynx which seems to be pathognomonic." Wood [31] said: "One of the most pathognomonic lesions of laryngeal tuberculosis is a smooth, reddened, swollen area, involving the vocal process and the region immediately above it."

Areas of hyperemia and pallor, any involvement of the interarytenoid sulcus and a congestion of one or both posterior thirds of the cords should lead one to suspect tuberculous involvement. Casselberry [37] considers interarytenoid hyperplasia pathognomonic. Certainly such a lesion should lead one to suspect tuberculous involvement.

Tuberculoma may be mistaken for a malignant condition; in fact, in some cases biopsy may be found necessary. In such an instance Wood [31] warned that the area from which the section is taken should be cauterized to prevent the dissemination of tubercle bacilli into the surrounding tissues. One must stress the danger that attends promiscuous removal of tissue for the study from the larynx. Greene [38] and Myerson [39] have seen cases that were activated by such a procedure.

A turban-shaped epiglottis or pear-shaped arytenoids are practically diagnostic. This is usually corroborated by the finding of involvement of the chest.

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36. Greene, J. B.: Laryngeal Tuberculosis, South. M. J. **7**:973 (Nov.) 1915.
37. Casselberry, W. E.: The Recognition of the Early Changes in the Larynx in Tuberculosis, J. A. M. A. **61**:1789 (Nov. 15) 1913.
38. Greene, J. B.: Personal communication to the author.
39. Myerson, M. C.: Personal communication to the author.

Freudenthal [40] emphasized the need of a laryngeal department in every tuberculosis sanatorium and a routine examination in every pulmonary case.

Thomson,[28] in his admirable treatise, commented "Of 477 cases no less than 5 per cent had no complaint about their throat, and their voices—both to their own ear and to mine—sounded quite clear." Undoubtedly these cases would not have come so early to the laryngologist's attention if symptoms had been awaited.

Spencer [21] stated: "A diagnosis should not be made from the laryngeal examination alone without the valuable aid of the internist, the roentgenologist, the pathologist and the bacteriologist." In the series of cases studied, this procedure was carried out. It is my opinion, however, that in a great majority of cases of tuberculous laryngitis a diagnosis can be made on the basis of what one sees.

When in doubt, repeated tests of the sputum should be made; conclusions must not be drawn from two or three examinations yielding negative results. In the 45 cases studied there were 10 negative sputums; of these negative sputums 4 were tested only once and 3 only three times, yet physical findings in the chest and roentgenograms showed every patient to have some degree of pulmonary involvement.

Differential Diagnosis.—Carcinoma must be eliminated as it occasionally resembles tuberculous laryngitis. It usually occurs in midlife or later, and is more frequently unilateral, while tuberculosis is generally bilateral. Carcinoma is a smaller and more localizing lesion, most frequently affecting the vocal cord. When granuloma is confusing, caution must be used in deciding on a biopsy, as already mentioned.

Syphilis, although prone to attack the anterior part of the larynx, may give a picture similar to tuberculosis. Every patient should have a Wassermann test of the blood. Four of the 45 cases presented in this study showed positive Wassermann reactions. A generalized edema of the larynx, such as is seen in syphilis, is much more pronounced than the lesion of tuberculosis. The syphilitic lesion destroys the identity of the structures; tuberculosis rarely does.

Chronic catarrhal laryngitis must be ruled out in some cases. It often precedes tuberculous involvement of the larynx, but responds to treatment more readily than tuberculosis. Greene [36] said of this condition: "There is a strong tendency for all the mucous membrane of the larynx to be equally affected and the vocal cords to appear evenly red and swollen. There is not seen the peculiar spindle shaped cord of one side so often observed in tuberculosis."

Other conditions less frequently met with are lupus, actinomycosis, pachydermia laryngis and leprosy.

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40. Freudenthal, W.: Prognosis and Treatment of Tuberculosis of the Larynx, Laryngoscope **22**:971 (July) 1912.

Cases, Lesions and Results

Case	Sex	Age	Wassermann Test	Sputum	Pulmonary Group*	Laryngeal Stage†	Interarytenoid Sulcus	Cords	Arytenoids	Aryepiglottic Fold	Ventricular Bands	Epiglottis	Subepiglottic Region	Tonsils	Gums	Ears	Results
1	M	51	—	—	III	C	..	+	+	..	..	..	+	..	..	..	Healed
2	M	66	—	+	III	C	+	+	+	+	..	..	..	..	..	..	Improved
3	M	25	—	+	III	SA	+	+	+	..	..	+	..	..	..	..	Worse
4	M	30	—	+	III	SA	+	+	+	..	..	..	..	..	..	..	Not improved
5	M	39	—	+	III	C	+	..	+	..	..	+	..	..	..	..	Not improved
6	M	36	—	—	III	C	+	+	+	..	..	..	..	..	..	..	Improved
7	M	31	?	+	III	A	+	+	+	..	..	..	..	+	..	..	Not improved
8	M	74	—	+	III	A	+	+	+	..	..	+	..	..	..	..	Worse
9	M	58	—	+	III	A	+	..	+	..	..	+	..	..	..	..	Not improved
10	F	44	—	+	III	C	..	..	+	+	..	..	..	..	..	..	Not improved
11	M	36	—	+	III	C	+	+	+	..	+	..	..	..	..	..	Healed
12	M	29	—	+	III	C	..	+	..	..	..	..	..	..	..	..	Improved
13	M	40	—	+	III	C	..	+	..	..	..	..	..	..	..	..	Worse
14	M	26	++++	—	II	C	..	+	+	..	..	..	..	..	..	..	Not improved
15	M	40	++++	—	III	C	+	+	+	+	..	+	..	..	..	..	Healed
16	M	30	++++	+	III	A	+	+	+	+	..	..	..	..	+	..	Worse
17	M	43	±	+	III	C	..	..	+	..	..	..	..	..	..	..	Improved
18	M	28	±	+	III	SA	+	+	+	..	+	+	..	..	..	..	Worse
19	M	24	++++	+	III	C	+	..	+	..	..	..	..	..	..	..	Improved
20	M	15	—	+	III	C	..	+	..	..	..	..	..	..	..	..	Healed
21	M	38	—	+	III	C	+	+	+	..	..	..	..	..	..	..	Improved
22	M	51	—	+	III	C	..	+	..	..	+	..	+	..	..	..	Improved
23	M	29	—	+	III	SA	+	+	+	+	+	+	..	..	..	..	Improved
24	M	46	—	+	III	A	+	+	+	..	..	+	..	..	..	..	Worse
25	M	36	—	—	III	SA	+	+	..	..	..	+	..	..	..	..	Not improved
26	M	27	—	—	III	C	..	+	..	..	..	..	..	..	..	..	Improved
27	F	42	—	+	III	A	..	+	+	..	..	..	..	..	..	..	Not improved
28	F	28	—	—	III	SA	..	+	..	..	..	+	..	..	..	+	Not improved
29	F	30	—	..	III	SA	+	+	+	..	+	+	..	..	..	..	Not improved
30	F	19	—	+	III	C	+	..	..	..	..	..	..	..	..	..	Not improved
31	F	26	—	+	III	A	+	+	+	..	+	..	..	..	..	..	Worse
32	F	31	—	+	III	SA	+	+	+	..	..	+	..	..	..	..	Worse
33	M	..	—	—	III	C	..	+	..	..	..	..	..	..	..	..	Improved
34	F	15	—	..	III	C	..	+	..	..	..	..	..	..	..	..	Improved
35	F	20	—	+	III	C	+	..	+	..	..	+	..	+	..	..	Improved
36	M	33	—	+	III	SA	+	+	+	+	+	..	..	+	..	..	Worse
37	M	28	—	+	III	SA	+	+	+	..	..	+	..	..	..	..	Not improved
38	M	37	—	+	III	SA	+	+	+	..	..	..	..	..	..	..	Worse
39	M	32	—	+	III	A	+	+	..	..	..	+	..	..	..	..	Not improved
40	M	28	—	..	III	C	+	+	+	+	+	+	..	..	..	..	Improved; tracheotomy
41	M	23	—	—	III	C	..	+	..	..	..	..	..	..	..	..	Improved
42	F	30	—	+	III	SA	+	+	+	..	..	+	..	..	..	..	Not improved
43	F	21	—	+	III	SA	+	+	+	+	..	+	..	..	..	..	Not improved
44	F	47	—	+	II	C	+	+	+	..	..	..	..	..	..	..	Not improved
45	F	21	..	+	III	C	+	+	+	..	..	+	..	..	..	..	Worse

* The stage of the pulmonary condition is given according to the Turban-Gerhardt classification of pulmonary tuberculosis. (Group I, slight involvement; group II, severe involvement; group III, advanced involvement.) In the official proceedings of the International Tuberculosis Committee the stages are more fully defined as follows: Group I, disease of slight severity limited to small areas of one lobe on either side; in case of infection of both apexes, it does not extend beyond the spine of the scapula or clavicle; in case of infection of the apex of one lung, it does not extend below the second rib in front. Group II, disease of slight severity, more extensive than in group I but affecting at most the whole of one lobe, or severe disease extending at most to half of one lobe. Group III, all cases of greater severity than those in group II and all those showing considerable cavities.

† A, acute; SA, subacute; C, chronic.

PROGNOSIS

The prognosis of laryngeal tuberculosis has changed considerably with the advent of the modern treatment of pulmonary and laryngeal lesions.

J. N. MacKenzie,[41] in writing of his experiences in 1879, said: "All were doomed to die; the appearance of the disease in the larynx was the warrant of death."

L. Gruenwald [42] stated that the cure of one (i. e., tuberculosis of the larynx) is impossible with the cure of the other (i. e., tuberculosis of the lungs).

Thomson [28] believes "tuberculosis of the larynx diminishes the hope of recovery from disease of the lungs by nearly one half."

Today the prognosis is not hopeless in all cases; in fact, many cures, over a period of years, are reported. Levy [43] said in referring to the prognosis, "I know of no disease into which so many items enter as into that of laryngeal tuberculosis."

In the acute and subacute forms the prognosis must be guarded. There is, however, some hope when these types assume the chronic form. In every case the extent of involvement in the larynx and in the lungs must be studied carefully and considered together. With an advancing lesion, either or both, the prognosis is unfavorable. It is interesting to note that the laryngeal condition can improve while the pulmonary disease increases. This occurred in 3 cases in this report.

The most favorable prognosis is offered in the chronic type in which the pulmonary involvement is of low grade or quiescent. Experience has shown that patients who present what I choose to call a disintegrating lesion are doomed, and a fatal prognosis is in order. Freudenthal [44] emphasized the poor prognosis in laryngeal tuberculosis occurring in pregnancy, and insisted on therapeutic abortion.

TREATMENT

The prevention of this most serious condition should first be considered. Dworetsky [29] advised: (1) breathing through the nose; (2) proper use of the voice; (3) restraint of cough, often unproductive; (4) prevention and prompt treatment of colds; (5) abstinence from tobacco and alcohol, and (6) examinations at frequent intervals. He

41. MacKenzie, J. N.: Some Reminiscences, Reflections and Confessions of a Laryngologist, Ann. Otol., Rhin. & Laryng. **25**:61 (March) 1916.

42. Gruenwald, L.: Some Observations in the Treatment of Laryngeal Tuberculosis, J. Laryng., Rhin. & Otol. **20**:637, 1905.

43. Levy, Robert: Laryngeal Tuberculosis, J. A. M. A. **60**:1518 (May 17) 1913.

44. Freudenthal, W.: The Baneful Influence of Pregnancy on Laryngeal Tuberculosis, Laryngoscope **17**:930, 1907.

expressed the opinion that the most important predisposing factor in laryngeal tuberculosis is the interference with normal breathing, owing to a greater or less degree of nasal obstruction, and he directs his prophylactic treatment to the nose. Most laryngologists at present are agreed that any infection of the sinuses or throat should be carefully treated.

Freudenthal [40] believes that all cough in tuberculosis is due to disease of the respiratory tract, particularly a catarrhal condition of the larynx, and consequently he advises attention to the postnasal drip.

Greene,[36] Terry [45] and others do not hesitate to perform tonsillectomies when indications are definite. Greene [36] stated: "In regard to the removal of diseased tonsils there can be no question of benefit to the patient in certain selected cases. They cause harm in two ways, first by being the site of repeated attacks of acute tonsillitis, thus lowering the patient's resistance, and in the second place causing a downward extension of infection, acute laryngitis." All operations on the nose and throat should be performed under local anesthesia.

Tonsillectomy must be cautiously approached and carefully considered in tuberculous laryngitis. All are agreed that this operation may be performed in carefully selected cases. Some have seen generalized miliary tuberculosis follow tonsillectomy. Myerson [39] saw two cases in which tonsillectomy was followed by general miliary tuberculosis and rapid decline in young adults. Fishberg [46] and Wood [47] are strongly opposed to this procedure.

The treatment of laryngeal tuberculosis should be undertaken with the laryngologist and phthisiologist working together. If the patient is steadily declining from the pulmonary disease, little can be accomplished in the larynx.

Wood [31] said: "In the pursuance of our local treatment, it is necessary to remember that the topical measures, either surgical or medical, should not be of a nature that tires or exhausts the patient, and it is sometimes wiser to dispense with the local treatment entirely, if it is followed by a rise in temperature and more or less exhaustion of the patient."

Thomson [28] considered that the treatment of the larynx "must be based upon the conception that it is but a complication of the original infection in the chest."

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45. Terry, G. H. B.: Eye, Ear, Nose and Throat Complications Occurring in Patients in a Hospital for the Tuberculous, Tri Sect. Laryng., Otol. & Rhin., A. M. A., 1929, p. 110.

46. Fishberg, M.: Pulmonary Tuberculosis, ed. 2, Philadelphia, Lea & Febiger, 1919, p. 517.

47. Wood, G. B.: Personal communication to the author.

Edson [48] and others are advocates of climatic treatment and believe that the patient has an advantage in high altitudes. The most effective palliative treatment is the use of mild oil sprays, such as 1 per cent menthol in liquid petrolatum, and the inhalation of tincture of benzoin compound.

Greene [36] suggested "that in all cases of laryngeal tuberculosis, particularly when the lesion is at the entrance to the larynx, the patient should abstain from the use of all irritating substances, such as oranges, lemons, grapefruit, and all peppery and highly seasoned food."

When pain exists, I have found that a 1 per cent aqueous solution of cocaine sprayed into the larynx just before meals will often give temporary relief. Orthoform or anesthesine lozenges may be of value in some cases. Narcotics, morphine and codeine, may have to be resorted to in cases of severe pain. In advanced cases with intractable pain much success has been met with by Fetterolf,[49] Vanderhoof [50] and others by injections of alcohol into the superior laryngeal nerve. Schugt [51] blocks the nerve by using a curved laryngeal needle introduced directly into the pyriform sinus. The relief in some cases is gratifying and may last from a few days to weeks.

The palliative and curative effects of chaulmoogra oil have brought the drug into the medical treatment of laryngeal tuberculosis. La Rue,[52] Lukens,[53] Van Poole [54] and Snapp [55] have reported their experiences. Lukens [53] stated: "At the present time, however (since the results of the local application of chaulmoogra oil, after considerable experimentation have been so gratifying), the use of alcohol injections in the Jefferson Hospital and the Phipps Institute have been reduced to an occasional one." I had the opportunity of working in Dr. Lukens'

48. Edson, C. E.: Climate in the Treatment of Laryngeal Tuberculosis, Ann. Otol., Rhin. & Laryng. **30**:888 (Dec.) 1921.

49. Fetterolf, G. E.: The Relief of Pain in Advanced Tuberculosis of the Larynx by Means of Injections of Alcohol into the Internal Laryngeal Nerve, Ann. Otol., Rhin. & Laryng. **21**:128, 1912.

50. Vanderhoof, D. A.: Technic and Results of Injections of Alcohol for Pain in Tubercular Laryngitis, Illinois M. J. **25**:139, 1914.

51. Schugt, H. E.: Superior Laryngeal Nerve Block Anesthesia Via Pyriform Sinus, Am. J. Surg. **7**:659 (Nov.) 1929.

52. La Rue, C. L.: Treatment of Tuberculous Laryngitis Considering Especially the Use of Chaulmoogra Oil, Tr. Am. Laryng., Rhin. & Otol. Soc. **34**:122, 1925.

53. Lukens, R. M.: Medical Treatment of Laryngeal Tuberculosis, in Jackson, C., and Coates, G. M.: The Nose, Throat and Ear, and Their Diseases, Philadelphia, W. B. Saunders Company, 1929, p. 882.

54. Van Poole, G. M.: Chaulmoogra Oil in the Treatment of Laryngeal Tuberculosis, Laryngoscope **40**:132 (Feb.) 1930.

55. Snapp, C. F.: Tuberculous Laryngitis Treatment with Chaulmoogra Oil, J. Michigan M. Soc. **26**:719 (Dec.) 1927.

clinic at the Phipps Institute, and saw favorable results. Some of the patients in this series studied were treated with chaulmoogra oil, but the results were not as satisfactory as I had hoped.

Vocal rest is one of the most important factors in the treatment of laryngeal tuberculosis; it diminishes discomfort, cough and pain. Thomson [28] reported that 23 of 67 patients on the silence treatment were cured. This procedure is best carried out in a sanatorium with intelligent patients. Silence should be prescribed in all acute cases and particularly in those showing lesions on the cords and interarytenoid sulcus and nearby regions where there is motion.

Whispering allows more freedom, and should be done with the lips so as not to bring the cords into apposition. Carmody [56] mentioned the fact that often more harm is done when a patient cannot whisper easily but strains, than when he is allowed to talk.

Many drugs have been applied locally to the laryngeal lesion with some measure of success. The galvanocautery has in a great measure taken their place with more satisfactory results.

Lactic acid, since its introduction by Krause [16] in 1885, has been widely used, but now has few enthusiastic adherents. This drug is supposed to penetrate the tissues and produces cicatrization. It is applied in an aqueous solution to the ulcerated area with a cotton swab, beginning with about 3 per cent and gradually increasing the strength to 20 or 30 per cent. A solution of formaldehyde is used in the same manner and strength, and is preferred to lactic acid by some laryngologists. Trichloracetic acid in strengths of from 3 to 30 per cent has been successful in some hands. The larynx must be cocainized prior to its use and the acid neutralized with a sodium bicarbonate solution after its application. The use of these drugs in some cases is quite objectionable to the patient.

Tuberculin has given good results in a few selected instances. It is injected into the larynx at the site of the lesion. Spencer [57] stated: "I have used this method of treatment, but cannot recommend it in preference to the cautery and other well established methods of treatment." Souchet [58] has reported favorable results by using Boquet and Nègre's methylated antigen of Koch bacilli injected subcutaneously.

Heliotherapy has for some time been a popular treatment in laryngeal tuberculosis, and holds its place as an effective therapeutic method.

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56. Carmody, T. E., in discussion on Terry (footnote 45).

57. Spencer, F. R.: The Medical and Surgical Treatment of Laryngeal Tuberculosis Including a Preliminary Report on the Direct Injection of Tuberculin into the Larynx, Ann. Otol., Rhin. & Laryng. **34**:424 (June) 1925.

58. Souchet: Tuberculous Laryngitis: Treatment with Boquet and Nègre's Methylated Antigen, Ann. d. mal. de l'oreille, du larynx **48**:900 (Sept.) 1929.

Sorgo,[59] in 1904, and recently Stafford,[60] Fischer,[61] Strandberg [62] and Ryan [63] reported their results and advocated its use. The solar laryngoscope which is used for this treatment consists of an arrangement of mirrors which reflect the sun's rays into the larynx. This can be carried out by the patient himself or by a nurse, but always under the guidance of the physician in order that the larynx will not be overtreated. Recently good results have been reported by Schreyer,[64] Moore [65] and others who are using the Wessely Radiation Machine, which is a modified carbon arc light that emanates artificial actinic rays. This method has a readily recognized advantage over sunlight. There is grave objection to subjecting the patient to direct laryngoscopy daily for such exposures as has been advocated.

Cauterization.—Cauterization has proved to be the best single method of treatment for laryngeal tuberculosis that is known today. Thompson,[28] Lockard,[18] Dean,[66] Levy,[67] Myerson,[39] Wood,[31] Spencer,[21] Iglauer,[68] Greene,[36] Freudenthal,[40] Dworetsky [34] and other prominent laryngologists advocate and strongly support the use of the galvanocautery. Spencer [21] stated: "It is a safe method when used carefully, and produces a surer, quicker and more permanent destruction of the tubercle than any other known method. However, like any other method of treatment, it cannot be used for all patients, nor should it be used for all patients suffering with laryngeal tuberculosis."

Use of the galvanocautery is rarely indicated in acute and subacute types of laryngeal tuberculosis. Patients with rapidly advancing pulmonary lesions, a high fever and frequent hemorrhages are not suitable

59. Sorgo, J.: On the Treatment of Laryngeal Tuberculosis with Reflected Sunlight, Wien. klin. Wchnschr. **17**:12, 1904.

60. Stafford, F. B.: Laryngeal Tuberculosis with Special Reference to Sunlight Treatment, Virginia M. Monthly **48**:239 (Aug.) 1921.

61. Fisher, E.: Heliotherapy in the Treatment of Tuberculous Laryngitis, U. S. Vet. Bur. M. Bull. **3**:1066 (Oct.) 1927.

62. Strandberg, O.: Heliotherapy and Artificial Light: Treatment of Tuberculous Conditions and Particularly Laryngeal Tuberculosis, J. A. M. A. **90**:1595 (May 19) 1928.

63. Ryan, M. C.: Tuberculous Laryngitis: Treatment with Reflected Sunlight, U. S. Vet. Bur. M. Bull. **6**:73 (Jan.) 1930.

64. Schreyer, W.: Treatment of Laryngeal Tuberculosis by Wessely's Carbon Arc-Light, Ztschr. f. Hals- Nasen- u. Ohrenh. **19**:430 (Feb. 28) 1928.

65. Moore, Roy S.: The Treatment of Laryngeal Tuberculosis, New York State J. Med. **26**:247 (March 15) 1926.

66. Dean, L. W.: Treatment of Tuberculous Laryngitis by Suspension Laryngoscopy, Ann. Otol., Rhin. & Laryng. **30**:898 (Dec.) 1921.

67. Levy, Robert: Surgical Treatment of Laryngeal Tuberculosis, Ann. Otol., Rhin. & Laryng. **30**:912 (Dec.) 1921.

68. Iglauer, S.: Electro-Cautery in Tuberculosis of the Larynx, Laryngoscope **26**:1237 (Oct.) 1916.

for cauterization. The general condition should be favorable, the pulmonary condition arrested or improving; this type responds best to galvanocauterization. The cautery then is used in chronic laryngeal tuberculosis. Myerson [39] stated: "Any lesion that lends itself to replacement fibrosis is a desirable one to treat with the cautery." Beginning superficial ulcers should be burned and infiltrates punctured, the arytenoid articulation being avoided if possible. The results are most promising when the lesions are on the cords, vocal process and interarytenoid space.

The preparation of the patient consists of the omission of the meal previous to the treatment. A sedative may be given a half hour before; Spencer [21] recommended from one-sixth to one-fourth grain (10.6 to 16.2 mg.) of morphine sulphate. In this series of cases, a sedative was seldom required. The larynx is anesthetized by applying a swab of 10 per cent cocaine solution containing a few drops of 1 : 1,000 epinephrine on a laryngeal applicator into each pyriform sinus or by dropping some of the solution about the rim of the larynx from a laryngeal syringe. Two or three applications are usually sufficient, and the patient is ready for cauterization in approximately five minutes.

Arrowsmith [20] and Dean [66] advocated cauterization under direct laryngoscopy. Myerson [39] and Thomson [28] prefer the indirect method. Thomson [28] wrote: "Treatment would, in most cases, do more harm than good if the applications were made under the more trying methods of direct or suspension laryngoscopy. I mention this as many of the younger school of laryngology have not taken the trouble to acquire the delicate art of manipulation under indirect laryngoscopy and thus limit themselves in all cases employing the easier (for them) but no more effective technique of the direct method." I have found that not only is the direct method disagreeable to the patient while he is being treated but he invariably complains of a sore throat afterward which interferes with eating for about three days. In my opinion this can be attributed to the pressure of the laryngoscope during the treatment. The indirect method of treatment offers no such possibility of trauma.

Infection or hemorrhage following cauterization is extremely rare. There is practically no after-treatment. Vocal rest should be adhered to if the area treated involves the more movable portions of the larynx. A mild oil spray may be used if desired. In most of the cases studied in this series, the treatment was carried out twice a month with from one to five punctures at a sitting according to the indications, the average number of treatments being three. A week or two is permitted to elapse, to allow the disappearance of any reaction incidental to the cauterization.

Of the 45 cases presented 12 were found suitable for cauterization. The results of treatment with the galvanocautery were as follows:

healed, 2; improved, 5; not improved, 4, and worse, 1. One of these patients had a beginning infiltration at the anterior commissure which spread to the cords. It was arrested in this unusual location by the cautery.

When the lesion is limited or nearly limited to the epiglottis and the usual methods of treatment are failing, amputation of the epiglottis has been advised. Most enthusiastic for this procedure were Lockard [69] and Arrowsmith [20] who reported the great relief that this operation gives to the intolerable odynophagia. It may arrest the disease temporarily, and in an occasional case a cure may result. It is not performed in hopeless cases in which the patients are rapidly losing ground. The operation consists of amputating the entire epiglottis with a special forceps in one bite or by using a cautery snare.

Tracheotomy.—Occasionally tracheotomy must be performed. It has been advised as a measure to place the larynx at rest, but this has had a small following. Tracheotomy is indicated when dyspnea threatens the patient's life owing to obstruction of the glottis by infiltration, granulomas or bilateral abductor paralysis. One patient of this series had to be tracheotomized. In this case both ventricular bands were greatly infiltrated, and a moderate sized granuloma presented from the interarytenoid space.

Pneumothorax.—Pneumothorax as a means of preventing and arresting tuberculosis of the larynx is receiving more and more attention. Thomson [70] has reported healing in 6 of 9 cases, and recently Dworetsky [71] reviewed the literature and reported his findings. His conclusions are:

1. A satisfactory pneumothorax, by arresting the pulmonary lesion, will almost always prevent the development of laryngeal tuberculosis.

2. A satisfactory collapse of the diseased lung by either artificial pneumothorax, phrenic avulsion or extrapleural thoracoplasty, usually exerts a beneficial effect on the laryngeal lesion.

CONCLUSIONS

The following are the conclusions drawn from this series of studies of laryngeal tuberculosis:

1. The treatment of laryngeal tuberculosis offers encouraging results in many or most early cases.

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69. Lockard, L. B.: Final Conclusions Regarding Amputation of the Epiglottis for Tuberculosis, Tr. Am. Laryng. A., 1917, p. 266.

70. Thomson, St. Clair: Tuberculous Laryngitis: Favorable Pneumothorax Healing Six of Nine Cases, Lancet **1**:963 (May 7) 1927.

71. Dworetsky, J. P.: Lung Immobilization in the Treatment of Pulmonary Tuberculosis and Its Influence on the Larynx (a Further Report), Ann. Otol., Rhin. & Laryng. **39**:1111 (Dec.) 1930.

2. It should be brought to the attention of the general practitioner that every patient with pulmonary tuberculosis should have a laryngeal examination at frequent intervals; one should not wait for laryngeal symptoms.

3. The treatment is best carried out in a sanatorium where the pulmonary and laryngeal condition may be under careful surveillance.

4. The most effective treatment today is electrocauterization, vocal rest and sunlight or modified sunlight.

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CEREBROSPINAL RHINORRHEA

A REPORT OF TWO CASES

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During the past year I have had two cases of cerebrospinal rhinorrhea, both of which were most instructive. One was spontaneous; one was traumatic. One patient is living; one is dead. Both presented points of interest; one perhaps contains a lesson, and I believe that both should be recorded. In the hope that they will prove of value, the history of each is presented herewith.

THE SPONTANEOUS CASE

History.—A woman, aged 60, was referred by Dr. John Ritchie on July 2, 1930. She was an unusually stout woman, weighing 225 pounds (102 Kg.); she had been obese since childhood, but had always enjoyed good health. In February, following an attack of influenza, she noticed a watery discharge from the right side of her nose. Recovery from the influenza was uneventful, and she resumed her household duties, but every morning awoke with a severe bitemporal headache. At 9 or 10 o'clock the headache disappeared, and the rhinorrhea began. The flow continued intermittently throughout the day and night, with the exception of the early morning hours, at which time headache was present.

The amount of fluid coming from the nose was about half an ounce (15 cc.) an hour, or approximately 12 ounces (355 cc.) a day. It slightly stained but did not stiffen the linen. The discharge was quite profuse, because each morning the pillow case was saturated. Bilateral pressure on the internal jugular veins occasionally produced a gush of fluid from the nostril.

Other nasal symptoms were absent, and there was no history of injury or operation.

Examination.—The first nasal examination showed that both passages were clear. The lining membrane was pale on each side. No secretions were visible, with the exception that with a nasopharyngoscope the right middle meatus was found to be moist. On transillumination, the frontal sinuses and the left maxillary were clear, while there was a haziness of the right maxillary. The sphenoethmoidal areas were normal. The ears, oral cavity, pharynx and larynx were normal.

Later examination showed the presence of an accessory maxillary ostium on the right side, and this opening, together with the larger normal one, was bridged by a film of watery secretion. Small doses of atropine, prescribed in the hope of

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checking the rhinorrhea, were ineffectual. The question of a catarrhal inflammation of the sinuses and especially the right maxillary was considered. Nasal hydrorrhea was excluded because the discharge was unilateral, continuous during sleep and always preceded by a temporal headache, which was relieved as the flow began. Consideration was given to aspergillosis of the maxillary sinus, but was dismissed because this rare condition is characterized by nasal stenosis, sneezing and a viscous discharge, none of which was present in this case.

The influence of posture was tried. With the head tilted slightly forward, the nasal secretion would appear on the upper lip. On leaning forward, with the head well flexed, large watery drops would emerge from the nose and fall to the floor. With the head brought back to the erect position and tilted to the right, the discharge would cease. When the head was tilted to the left, the discharge would reappear. These findings led to the suspicion of antral pathologic changes, but it was difficult to find any clinical entity to which a discharge of this character and chronicity could be attributed.

A roentgenographic study of the paranasal sinuses made by Dr. E. P. Pendergrass showed a clouding of the right maxillary, while the other sinuses were clear. Lavage of the suspected antrum was negative, the fluid returning clear. No culture was taken. A second roentgenography, by Dr. Pendergrass, after an injection of opaque oil, revealed only a slight thickening of the lining membrane.

The clinical findings of a film over the ostium of the right maxillary sinus, the haziness on transillumination, the distinct influence of posture and the roentgenographic report of right maxillary cloudiness only proved to be misleading, because later it was shown by placing a cotton tampon between the middle turbinate and the septum that the discharge had its origin in the superior meatus. After several examinations failed to disclose the source of the fluid and with the evidence pointing toward the superior group of cells, a new line of thought was opened, and a tentative diagnosis of cerebrospinal rhinorrhea was made. The patient was then referred to the service of Dr. George Fetterolf at the University of Pennsylvania Hospital, where she was placed under my care. Examination of the blood, roentgenographic studies and various consultations were then made.

On several occasions the systolic blood pressure read 150 mm. of mercury; the diastolic, 95 mm. The hemoglobin was 95 per cent; the red blood cells numbered 4,640,000; the white blood cells, 8,300, with polymorphonuclear leukocytes, 78 per cent; lymphocytes, 18 per cent; mononuclears, 3 per cent, and eosinophils, 1 per cent. The blood sugar was 112 mg. per hundred cubic centimeters; the urea nitrogen, 26 mg. The Wassermann reaction of the blood was negative. Urinalysis of three specimens gave negative results. The basal metabolic rate was within normal limits.

The nasal discharge was collected in a sterile test tube and sent to the laboratory for study. Dr. Wendall C. Hall gave the following report: "A watery, slightly turbid fluid which did not foam readily; specific gravity, 1.005; reaction, alkaline; no precipitate on boiling; no reduction of Fehling's solution with 3 cc. of fluid; however, a subsequent examination by the colorimetric method proved sugar to be present in the amount of 102 mg.; protein 54 mg. per 100 cc.; cell count 2-5 per c.mm.; a faint blue color was obtained by adding a trace of copper sulphate in excess of caustic potash; cold nitric acid gave a faint white precipitaate which disappeared on heating and reappeared on cooling." Dr. Hall's opinion was as follows: "The fluid is in the nature of a transudate and most certainly does not have the characteristics of an exudate. In view of all the above tests, this specimen could well be cerebrospinal fluid."

The roentgenographic study of the intracranial contents by Dr. E. E. Pendergrass revealed the following: "There is a peculiar appearance of the middle fossa, identity of which is uncertain. We cannot exclude a growth in this region. The pituitary fossa is within normal limits of size and there is no evidence of any convolutional markings."

Dr. W. E. Fry gave the following ophthalmologic report: "Media clear, disks round, margins well defined, vessels normal, macula and periphery negative. The interior of the eyes does not show any pathological change. There is a slight contraction of the visual fields for form."

Neurologic examination by Dr. A. M. Ornsteen did not elicit any finding to indicate the presence of an intracranial neoplasm. It was suggested by Dr. Ornsteen that spontaneous decompression of a hydrocephalus explains some cases of cerebrospinal rhinorrhea.

In an effort to determine definitely the point of exit of the fluid from the cranial cavity, it was decided to inject a dye into the spinal canal and then watch for its emergence into the nose. Preparations were made for a manometer reading and the injection of methylene blue (methylthionine chloride, U.S.P.). However, because of the patient's fat back all attempts to enter the spinal canal were unsuccessful; so the thought of exactly locating by this method the point of exit of the discharge had to be abandoned.

A curious sequel followed these attempts. In the afternoon, when the patient complained of lumbar pain, two tablets of a barbital derivative were given, and in the evening, owing to her restlessness, she received a hypodermic injection of morphine sulphate, one-fourth grain (0.016 Gm.). The following morning the nurse had difficulty in arousing her at 5 o'clock. Two hours later, however, she was fully conscious and moved about slowly in bed, but was slightly cyanotic. At 9:00 a. m., there was a sudden grave change manifested by extreme cyanosis, pulmonary edema and coma. There was no contraction of the pupils. The pulse was rapid and weak, but the blood pressure and temperature were normal. The pillow case was dry, and nasal examination did not show any secretion. To combat her immediate danger, large doses of atropine and caffeine were given, but at 10:30 o'clock the respirations had dropped to 10 per minute and remained at this alarming level for two hours. It is significant that with this drop in respiration the pulse became slower but of better volume and the temperature receded below normal.

It was naturally felt that the patient's grave condition was due to a cessation of the rhinorrhea, which had caused an increase in the intracranial pressure. Dr. Fetterolf suggested that decompression be tried by shrinking the upper nasal mucosa. With a Ballenger submucous elevator, the middle turbinate was displaced laterally and a large epinephrine-soaked tampon inserted high in the superior meatus. After ten or fifteen minutes the tampon was removed, and a search with the nasopharyngoscope brought the face of the sphenoid sinus into view. Oozing out of its ostium could be seen a clear watery fluid. Later, with the patient in a semirecumbent position and the head tilted forward, examination of the sphenoid revealed a clear discharge pulsating synchronously with the heart beat.

At 12:30 p. m. the respirations increased to 12 per minute, the cyanosis diminished, and the patient moved her right arm, opened her eyes and mumbled a few words. From that time consciousness gradually returned, and she began to recognize members of her family.

One might well ask why decompression failed to occur on this particular night. Two explanations occurred to me. One was that the tablets of a barbital derivative and the morphine sulphate induced such profound sleep that the normal changes

in the position of the head were prevented, and hence the usual postural decompression was not effected. The other was that the severe reaction was due alone to the narcotic influence of the drugs given the patient and had no relation to any increased pressure within the cranial vault.

It is now two years since the onset of the rhinorrhea, and the patient is at present attending to her household duties. Many drugs, including atropine, potassium iodide, benzyl benzoate and thyroid extract, have been tried to alleviate her symptom, but the only therapy of proved value is a reduction in the amount of fluid intake.

A roentgenogram taken a few weeks after the attack of pulmonary edema and coma tended to substantiate the clinical discovery of the sphenoid origin of the rhinorrhea. The report given by Dr. Karl Kornbloom read as follows: "There has been no change in the appearance of the pituitary fossa since the last examination. The dorsum sella is quite thin as if it may have been eroded from behind. This may also have caused an erosion into the sphenoid sinus and would thus account for the cerebrospinal rhinorrhea."

Historical Data.—Charles Miller,[1] in 1826, described the first definite case of cerebrospinal rhinorrhea in a boy with chronic hydrocephalus. Autopsy showed "an opening above and to the right of the crista galli which might have admitted four bristles and had a direct communication with the nose." The rhinorrhea lasted two years, and the boy died at the age of 16 of a complicating meningitis. A description of a clear nasal discharge probably of cerebrospinal origin is found in the writings of both Willis[2] in 1676 and Morgagni[3] in 1762. In 1877, Tillaux[4] reported a case that was the first to be verified by a chemical examination of the fluid. A valuable monograph of Sir St. Clair Thomson[5] appeared in 1899, in which he recorded twenty cases and described one of his own. During the next quarter of a century twenty more cases were reported. These were summarized by Johnston[6] when he recorded his case in 1926.

In review of the literature of the past five years, twenty-seven cases have been found, which brings the total number, spontaneous and traumatic, to sixty-nine.

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1. Miller, Charles: Case of Hydrocephalus Chronicus, with Some Unusual Symptoms and Appearances on Dissection, Tr. Med.-Chir. Soc. Edinburgh **2**:243, 1826.

2. Willis, Thomas: Cerebri anatome nervorumque descriptio et usus: quibus accesit viri cuiusdam clarissimi de ratione motus musculorum tractatus singularis, Opera omnia, Geneva, S. de Tournes, 1676.

3. Morgagni, J. P.: De sedibus et causis morborum per anatomen indagitis libri quinque, Venice, ex typog. Remondiniana, 1761, book 1, epistle 15, art. 21.

4. Tillaux, P. J.: Traité d'anatomie topographique avec applications à la chirurgie, Paris, Asselin & Cie, 1877, p. 56.

5. Thomson, St. Clair: The Cerebrospinal Fluid: Its Spontaneous Escape from the Nose, London, Cassell & Co., 1899.

6. Johnston, W. H.: Cerebrospinal Rhinorrhea, Ann. Otol., Rhin. & Laryng. **35**:1205, 1926.

The most exact knowledge as to the cause of spontaneous cerebrospinal rhinorrhea has been contributed by Locke,[7] who in 1926 searched the literature and found fourteen fatal cases in which necropsy had been performed. Among these reports, those of Paget and Baxter were too incomplete to be of value. In each of the remaining twelve studies internal hydrocephalus was found, which confirmed the opinion of Leber [8] expressed in 1883. Eight of the twelve cases showed a cerebral tumor producing an obstruction to the ventricular system. Two were due to congenital hydrocephalus, and two were cases of hydrocephalus of the adult type.

Sir James Barrett,[9] also in 1926, contributed a case having a bilateral tumor of the acoustic nerve as the etiologic factor. Recently Friedman's [10] patient with cerebrospinal rhinorrhea had a pituitary tumor removed. Autopsy did not show any connection between the pituitary fossa and the nose. A tumor in the region of the pituitary extending through the floor of the hypophysis and invading the left sphenoid sinus was reported in 1930 by Roberts.[11] This unusual case had an associated acromegaly with typical ocular and roentgen findings of a chiasmal lesion.

Bregman [12] and Redlich,[13] German authors, pointed to the significance of an erosion of the sella turcica, which in their opinion explains the frequency of rhinorrhea in tumors of the pituitary gland. Mankowsky [14] claimed that most tumors of the pituitary with destruction of the sella do not show rhinorrhea. He contended that in the majority of cases of rhinorrhea a connection could be shown between the anterior cranial fossa and the nasal cavity through the lamina cribrosa. This opinion bears out the findings collected by Locke, who in eleven of twelve cases found unmistakable openings connecting the floor of the anterior cranial fossa with the nasal cavity. Following the pioneer

7. Locke, C. E., Jr.: The Spontaneous Escape of Cerebrospinal Fluid Through the Nose: Its Occurrence with Brain Tumor, Arch. Neurol. & Psychiat. **15**:309 (March) 1926.

8. Leber, T.: Ein Fall von Hydrocephalus mit neuritischer Sehnervenatrophie und continuirlichen Abträufeln wässeriger Flüssigkeit aus der Nase, Arch. f. Ophth. **29**:273, 1883.

9. Barrett, James: Cerebrospinal Rhinorrhea, M. J. Australia **2**:182, 1926.

10. Friedman, A. P.: Tumor hypophysis et liquorrhoea nasalis, Arch. f. Psychiat. **88**:358, 1929.

11. Roberts, B. F.: Laryngoscope **40**:855, 1930.

12. Bregman, L. E.: Ueber den spontanem ausfluss von Cerebrospinalflüssigkeit durch die Nase, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **15**:474, 1907.

13. Redlich, E., cited by Mankowsky.[14]

14. Mankowsky, B. N.: Zur Frage über spontanem Liquorabfluss bei Hirntumoren (Liquorrhea nasalis), Ztschr. f. d. ges. Neurol. u. Psychiat. **119**:514, 1929.

work of Key and Retzius,[15] Locke showed that when celloidin masses were injected under pressure into the subarachnoid spaces of the dog, they frequently leaked from the nose. His belief was that the dura overlying the cribriform region was especially susceptible to penetration, because of the tiny holes that are normally present for the exit of the olfactory nerves; furthermore, the arachnoid membrane and the subarachnoid space continue for a distance along these nerves.

Loftus[16] suggested that the mode of exit of the fluid from the cranial cavity was through the remains of the embryonic craniopharyngeal canal. He thought that this canal might be forced open by a sudden increase in the intracranial pressure, such as in coughing, sneezing or laughing. Johnston in a later report expressed belief in this pathway.

Comment.—The spontaneous case reported here shows an exceptional route of the cerebrospinal fluid into the nasal cavity. By thoroughly shrinking the mucosa of the middle turbinate and passing the nasopharyngoscope, the discharge could be seen at the right sphenoid ostium. Since the presentation of this paper Smith and Walter[17] have reported a case of a cyst of the pituitary body with a complicating cerebrospinal rhinorrhea. They suggested the possibility of the sphenoid origin of the discharge, but the exact fistulous tract could not be seen.

A definite opinion concerning the actual intracranial pathology is not possible, but in the light of the experiences of Locke and others in finding unsuspected tumors of the brain producing spontaneous cerebrospinal rhinorrhea I am inclined to classify this case in the same category. Possibly there is a midline tumor in the region of the third ventricle with the rhinorrhea acting as spontaneous decompression.

THE TRAUMATIC CASE

History.—In February, 1931, a man, aged 57, was referred from the medical outpatient department of the Misericordia Hospital to the service of Dr. John E. Loftus. This patient had had bronchial asthma for more than twenty years. The attacks occurred in the early morning and were invariably aggravated by any climatic change. There was a history of a chronic infection of the paranasal sinuses and a spasmodic productive cough. The family history was irrelevant with the exception that one daughter had hay fever. Allergic studies were made by the medical service, but they failed to reveal any specific sensitivity. A culture was taken from the material secured by bronchoscopic drainage, and a vaccine was made, but the serum therapy gave no relief.

15. Key, E. A. H., and Retzius, G.: Studien in der Anatomie des Nervensystems und des Bindegewebes, Stockholm, Samson & Wallin, 1875-1876.

16. Loftus, J. E.: Cerebro-Spinal Rhinorrhea with Report of a Case, Laryngoscope **33**:617, 1923.

17. Smith, Carroll, and Walter, Lorenzo: Cerebrospinal Rhinorrhea with Cyst of the Pituitary Body, Arch. Otolaryng. **14**:610 (Nov.) 1931.

Examination.—Physical examination showed a tall robust man of ruddy complexion, weighing 177 pounds (80.3 Kg.). The pulse was regular and of good volume. His heart sounds were distinct, and no murmurs were heard. The systolic blood pressure was 150 mm. of mercury; the diastolic, 90 mm. The chest was resonant throughout; expirations were prolonged, and wheezing râles were audible. The abdomen and extremities were normal. Urinalysis did not show any albumin or sugar. The blood picture was as follows: red cells, 4,200,000; white cells, 7,300; hemoglobin, 90 per cent. The coagulation time was five minutes.

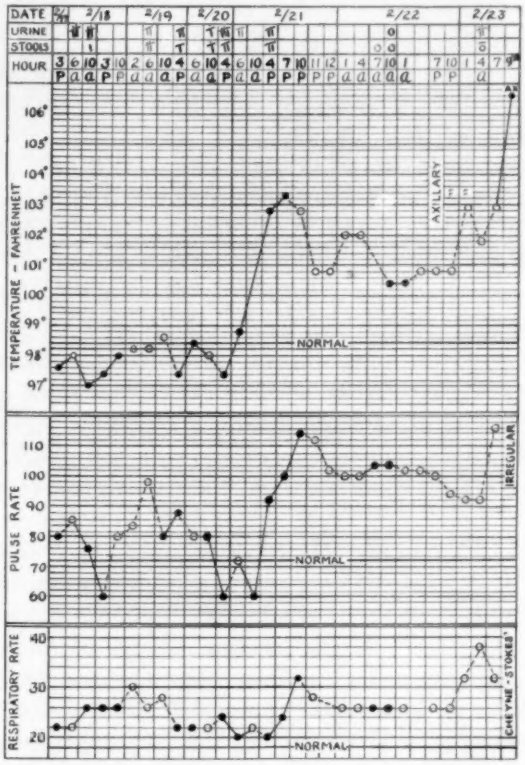
Nasal examination showed advanced hyperplasia of both middle turbinates and the presence of polypi in the middle meatuses and the left spheno-ethmoidal area. The right spheno-ethmoidal region was normal. On transillumination, the frontal sinuses and the right maxillary were hazy. The left maxillary was clouded. A roentgenographic study by Dr. E. J. Bertin revealed the following: "The frontal sinuses are extremely small but clear. The ethmoids and sphenoids show definite evidence of disease. The right antrum appears normal while the left one is almost totally obscured by disease."

As it was thought that the sinus pathology was an important complicating factor of the asthma, the patient was admitted to the hospital on February 17 for a bilateral ethmoidectomy. Two hours before operation on February 18, 6 grains (0.4 Gm.) of sodium amytal were given, and the operation was started about 3 p. m.

With the patient in a semirecumbent posture and the head on a horizontal plane, the ethmoidal areas were anesthetized with a 10 per cent cocaine-epinephrine solution. When the right middle turbinate was removed, extensive polypoid degeneration was found in the posterior ethmoidal cells. With a Blakesley alligator forceps some superficial tissue was removed, and the second bite into this polypoid mass was followed by a thin watery discharge. I suspected this to be cerebrospinal fluid, especially after further observation showed the leakage becoming more profuse and the fluid pulsating. Operation was stopped at once and the patient sent to bed. While he was being placed on the ward carriage, he fainted. On the basis that this was due to a sudden decompression and that the loss of fluid was too great, the ethmoidal area was packed with iodoform gauze. Epinephrine and atropine were given as stimulants, and in a few minutes the patient regained consciousness. When he returned to the ward he was cold and clammy, and he complained of temporal headache. His temperature was 97.2 F., and his pulse rate was 60. Hot bottles were applied to combat shock. Dr. Loftus was called in consultation and advised removal of the nasal packing and a policy of dehydration in order to reduce the intracranial pressure and perhaps facilitate an early closure of the operative wound. To this end fluids were restricted, and large doses of magnesium sulphate were given during the next twenty-four hours.

On the evening of operation the temperature was normal. The patient complained of a slight chill, but otherwise was comfortable. The next morning he felt a continual dripping in his throat, and when he leaned forward a watery discharge came from the right nostril. Unfortunately a specimen was not collected. No attempt was made at intranasal examination, but the oropharynx was perceptibly moist. Later in the day, or about twenty-four hours after operation, the rhinorrhea stopped, and no untoward symptoms were present. We felt, however, that because of the danger of meningitis the patient would require close observation for several days. On February 20, two days following the operation, his temperature and pulse remained normal (see chart) and his strength was such that he repeatedly requested to go home. We were quite happy in believing that he had weathered the storm and all danger had passed. On February 21, seventy-two

hours after operation, his temperature soared to 103 F., and he complained of severe occipital headache, chills and nausea. He was very restless and soon became delirious. His pupils were contracted, but reacted to light. Five and one-half hours after the onset of these symptoms, a spinal puncture produced 5 cc. of turbid fluid, but the pressure was not increased. The cell count was 16,000 polymorphonuclears. A later report showed hemolytic streptococcus in pure culture. On the following day, his headache was intense, and his restlessness and delirium increased. Kernig's sign and cervical rigidity were present. Spinal puncture now showed a cloudy fluid under pressure, and 35,000 cells were found. At 7 o'clock the next



Curves of the temperature and pulse while the patient was in the hospital.

morning he became stuporous, and his pupils were fixed. The labored respirations assumed a Cheyne-Stokes character, and the patient died at 10 a. m., five days after operation.

Autopsy.—The autopsy report by Dr. Max Strumia was as follows: The top of the skull was opened, and a large amount of thick purulent cerebrospinal fluid was found. Culture from the meninges showed Streptococcus haemolyticus. All of the brain surface was covered by a thick, yellowish, purulent exudate, which was especially abundant toward the base. The convolutions were flattened. After removing the brain, the base of the skull was carefully studied. An opening with ragged edges, measuring about 1 by 1.5 cm., was found on the right ethmoidal roof

1 cm. from the midline, or 1 cm. from the lamina cribrosa. This opening led into the ethmoid cells, which were filled with a soft, foul-smelling, gelatinous mucosa. The corresponding area on the orbital extension of the left ethmoidal roof was formed by extremely thin bony plates which could be easily broken or torn away with slight traction. There appeared to be senile atrophy of the bone. No necrosis was found.

Historical Data.—Apparently the escape of cerebrospinal fluid is a rare complication, because Campbell,[18] in 1928, reviewed the literature and found only three cases directly attributed to operations on the ethmoid sinuses. He added another case following ethmoidectomy and commented that the complication was not as serious as is commonly believed. A case reported by Cushing [19] in 1921 was probably the first one of postoperative rhinorrhea to be reported. This followed a sphenoethmoidal drainage. The discharge did not subside until a tumor of the brain was removed one year later. Autopsy disclosed a defect in the left ethmoidal region through which there was a dural protrusion. Cushing concluded that owing to the long-standing hydrocephalus due to the subtentorial tumor the cranial bones had undergone some absorption, and a pull on the wall of one of the ethmoid cells dislodged a piece of the plate through which the hernia occurred.

Hunt [20] devised an ingenious but formidable operation to combat the onset of meningitis. After opening the frontal sinus and removing the posterior wall, he elevated the dura and found the perforation of the ethmoidal roof. This was walled off by iodoform gauze, and after a week the skin flap was reopened and the packing removed. Recovery was uneventful.

The patient whose case was reported by Alden [21] responded successfully to the conservative treatment of dehydration and rest. The rhinorrhea lasted a week. In Campbell's case the discharge stopped in twenty-four hours. He advised a policy of increasing fluids immediately after operation in the hope that the discharge would cleanse the infected area. The Bulsons [22] have observed a case of rhinorrhea following a turbinectomy, the discharge lasting six years without any ill effects.

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18. Campbell, E. H.: Cerebrospinal Rhinorrhea Following Intranasal Surgery, Ann. Otol., Rhin. & Laryng. **37**:865, 1928.

19. Cushing, Harvey: Further Concerning the Acoustic Neuromas, Laryngoscope **31**:208, 1921.

20. Hunt, John G.: Accidental Rupture of the Ethmoidal Roof with Subsequent Recovery, Arch. Otolaryng. **3**:452 (May) 1926.

21. Alden, A. M.: Cerebrospinal Fistula Following Ethmoidectomy: Recovery, Ann. Otol., Rhin. & Laryng. **36**:121, 1927.

22. Bulson, A. E., and Bulson, E. L.: Cerebrospinal Rhinorrhea Following Intranasal Surgery, J. A. M. A. **93**:1969 (Dec. 21) 1929.

Comment.—The tragic outcome in this case recalls again the danger of surgical intervention on the ethmoid. Despite the scarcity of reports of meningitis and death following a cerebrospinal leakage during an ethmoidal operation, I believe that the immediate complication, per se, is not extremely unusual, but has probably passed unnoticed. The watery discharge, sometimes tinged with blood, could easily be construed as part of the postoperative reaction, and when the rhinorrhea lasts only twenty-four hours it may be overlooked. If the discharge is confined to the postnasal region, one might have no knowledge of its presence.

The autopsy in the postoperative case reported here proves that the ethmoidal roof was perforated and that the corresponding orbital roof was extremely thin and atrophic. This would favor the opinion that only a moderate amount of traction on the hyperplastic tissue was necessary to break down its bony attachment with a rent in the dura and a subsequent leak of fluid. This thinness and atrophy of the bone are an explanation but not an excuse for the accident. Every nasal operator should approach the paranasal sinuses on the basis that they are thin-walled and should be handled as such.

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A NEW TEST FOR THE DIAGNOSIS OF DISEASE OF THE INNER EAR

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That the auricles of both human beings and animals show involuntary movement on acoustic irritation has been established in the otologic literature since the beginning of the last century. Known as Preyer's reflex, since Preyer [1] revealed that the striking of tuning forks caused an involuntary jerking reflex of the auricle in new-born guinea-pigs and young rats, this phenomenon has been noted time and again, and is constantly employed in physiologic experimentation on animals as a proof that the animal hears. This reflex movement has also been discerned in man, and is described in the older as well as in the newer handbooks on otology. Lincke [2] and Politzer [3] frequently found patients having pathologic conditions in the ear who showed unconscious movements of the auricle either in a single circumscribed sector or in toto. Similar observations were mentioned by Urbantschitsch [4] and Lucae.[5] Schaefer and Giesswein [6] and Cemach [7] reported involuntary movements of the auricle during the process of listening. It must be said that prior to our own investigation we were unable to find any written comment, either in German or in English sources, on a practical clinical utilization of the reflex movement in man.

From the ear department of the Wiener Allgemeine Poliklinik, service of Professor Gustav Alexander.

1. Preyer, T. W.: Die Seele des Kindes, Leipzig, T. Grieben, 1890, p. 72.
2. Lincke, C. G.: Handbuch der theoretischen und praktischen Ohrenheilkunde, Leipzig, J. C. Hinrichs, 1837, vol. 1, p. 444.
3. Politzer, A.: Lehrbuch der Ohrenheilkunde, ed. 5, Stuttgart, F. Enke, 1908, p. 44.
4. Urbantschitsch, V.: Lehrbuch der Ohrenheilkunde, ed. 5, Berlin, Urban & Schwarzenberg, 1910, p. 485.
5. Lucae: Die chronische progressive Schwerhörigkeit ihre Erkenntnis und Behandlung, Berlin, Julius Springer, 1907, p. 360.
6. Schaefer and Giesswein, in Denker, A., and Kahler, O.: Handbuch der Hals- Nasen-Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 6, p. 518.
7. Cemach: Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **14**:871, 1924; Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1924, vol. 1, p. 871.

For purpose of clarity we have called the involuntary response of the auricle, whether partial or total, to tuning forks the "auricle reflex." Our observations covered a total of 198 cases. Among the subjects were included patients with normal hearing, with disease of the upper respiratory tract but negative findings in the ear and with external otitis, eczema, cerumen, acute and chronic catarrh of the middle ear, acute and chronic otitis, former simple and radical operations, cochlear and vestibular lesions, deafness, deaf-muteness and otosclerosis. In the 198 cases the reflex was definitely positive in 55. The 55 patients with positive reflexes had either cochlear and vestibular lesions or involvement of the middle ear with secondary lesions of the inner ear. As an illustration of our positive findings, we report several characteristic cases.

REPORT OF CASES

CASE 1.—M. H., aged 30, unemployed, on otoscopic examination showed scars on both ear drums. The Rinne test was negative, bone conduction was shortened, and the whispered voice was heard at one-half meter. On turning there was no reaction. Supplementary findings were an interstitial keratitis, Hutchinson's teeth and a pscudofistula reaction. The auricle reflex was found in both ears. With a slight vibration of the *C* 1 and *C* 4 forks, both right and left auricles swung in and out for from three to four minutes in constant waves. With a strong vibration of the tuning forks nothing occurred. With a medium vibration there was at first no response momentarily; then the reaction began and lasted for a long time. The helix was especially involved in the undulating movement.

CASE 2.—P. N., aged 64, a housewife, showed a combination of lesions of the middle and the inner ear. A radical operation had been performed on the right ear. The findings were: a negative Rinne test, lengthened bone conduction and a shortened time of hearing high sounds. The left ear drum was opaque and the Rinne test positive with shortened bone conduction and a shortened response to high sounds. The response to the fork was shortened ten seconds. Spontaneous movement was observed in the right ear. With a loud striking of the *C* 1 and *C* 4 forks, the auricle reflex was elicited. Movement of the middle parts of both the right and the left auricles took place. The movement was more pronounced in the left ear.

CASE 3.—E. G., aged 21, unemployed, had had an attic perforation in the right ear and a radical operation on the left. In the right ear, the Rinne test was negative, the whispered voice was heard at one-half meter, bone conduction was shortened and the response to the *C* 4 fork was shortened twenty seconds. In the left ear, the whispered voice was heard at 2 meters, bone conduction was lengthened and the response to the *C* 4 fork was shortened ten seconds. After several repeated trials with the *C* 1 fork, the auricle reflex manifested itself in the form of a definite movement of the upper half of the right auricle. No movement was observed with the *C* 4 fork. The left auricle responded similarly but in a weak fashion. No spontaneous or voluntary movements could be seen.

CASE 4.—J. R., aged 37, a barber, had a negative Rinne test with the bone conduction lengthened and the hearing of high sounds shortened by twenty seconds. The whispered voice was heard by the right ear at 3 meters and by the left ear at one quarter of a meter. No spontaneous movement of the auricles was present.

With a moderate striking of both the *C* 1 and *C* 4 forks there was a marked waving, rhythmic movement of both auricles. When the upper part of the auricle moved outward the inner part moved inward, and vice versa. No voluntary movements were seen.

Case 5.—L. R., aged 51, with no occupation, had a combination of lesions of the middle and the inner ear. Otoscopically there were a chronic adhesive process in the right ear and a dry perforation in the left. The whispered voice was heard by the right ear at 1 meter and by the left ear at one quarter of a meter. The Rinne test was negative. The response to the *C* 4 fork was shortened fifteen seconds in the right ear and thirty-five seconds in the left. The auricle reflex manifested itself by brisk movements in both auricles, particularly the left, with the *C* 1 fork, the low struck *C* 4 fork and the high struck *C* 4 fork. When either fork was held before the right auricle there was an associated movement in the left auricle. The reverse phenomenon was also seen. No spontaneous movement was noted, but there was a definite voluntary movement.

Case 6.—I. M., aged 48, a housewife, had a lesion of the right inner ear. The Rinne test was positive, with bone conduction shortened and the response to the *C* 4 fork shortened six seconds. Some spontaneous movement was present. There was definite movement of the lower half of the left auricle only with the *C* 1 fork. The auricle reflex was absent in the right ear.

Case 7.—J. G., aged 45, unemployed, showed a lesion of the left inner ear. Otoscopically there were scars on the drum. The whispered voice was heard by the right ear at 8 meters and by the left at 6 meters. The Rinne test was positive; the Schwabach test and response to the high sounds showed that the right ear was normal, while the response of the left ear showed that the time of hearing was shortened. The auricle reflex was positive. Movement of the middle part of the left auricle occurred with the *C* 1 fork but not with the *C* 4 fork. The auricle reflex was absent in the left ear. The patient had a generalized muscle reflex with the high *C* 4 fork, as well as a marked auropalpebral reflex.

Case 8.—M. S., aged 44, a housewife, had an involvement of the left inner ear. Otoscopically both drums were normal. In the left ear both the bone conduction and the response to the *C* 4 fork were shortened. With the *C* 1 and *C* 4 forks there was a slight movement of the middle part of the left auricle, elicited best when the forks were drawn to a distance. In the right ear a questionable faint movement occurred with the *C* 1 fork.

Our examinations showed that, in response to tuning forks, the auricle reflex is not rare and has some diagnostic significance. It is a rhythmic jerking of a part or of the whole of the auricle, in and out or up and down; sometimes the reaction is only of the helix, occasionally of the lobule of the auricle. Infrequently there are gross excursions of the auricle and more often small excursions. Often the movement followed a weak striking of the tuning forks. The most marked movement was obtained with the middle *C* 1 fork rather than with the high *C* 4 tuning fork.

The auricle reflex can be produced by holding the tuning fork ad concham or by placing it over the patient's forehead or mastoid prominence. The observer must be careful to employ bright daylight, so as to eliminate any possibly confusing shadows. Other pitfalls to be dis-

counted are movements of the auricle synchronous with breathing, pulsation of the veins of the neck, chewing, talking and voluntary facial movements. In some cases we saw a preexistent, although unconscious, spontaneous movement of the auricle accentuated vividly when the tuning fork was held ad concham. In another group of cases a tuning fork held before one auricle induced a corresponding movement in the fellow auricle.

In the cases in which the reflex was positive an involvement of the inner ear was most frequently found; less often, a combination of lesions of the middle and the inner ear. In one case with a positive reflex the ear was normal, while in one other case an acute serous catarrh was the underlying pathologic process. If the cochlear nerve is affected, the auricle reflex is more manifest than when combined lesions are present. No movement was to be seen in lesions of the middle ear associated with poor hearing (otosclerosis). The auricle reflex occurs more often when the hearing is poor. In unilateral cochlear lesions there is either no movement to be seen or only slight movement. We were unable to observe any movement in cases of complete deafness.

The reflex appears in cases with slowly progressive lesions of the cochlea. These patients must listen more attentively to sounds than persons with normal hearing who hear without straining. Consequently, the added effort produces a more intensive tonus of the muscles of the auricle. The increased attentiveness of the patient resolves itself as a movement of the expectant auricular muscles which, in turn, evince an increased tonus. Ostmann,[8] Lucae,[5] Urbantschitsch [4] and Schaefer and Giesswein [6] found the auricular reflex almost always during weak sounds, even on the threshold of hearing.

CONCLUSIONS

Our observations show that the auricle reflex can be used for clinical diagnosis. If the findings are positive, involvement of the cochlear nerve is suspected, more often primary than secondary. Of course, if the reflex is absent, a lesion of the inner ear cannot be excluded.

8. Ostmann, quoted by Lucae (footnote 5).

HEARING BY BONE CONDUCTION

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Is the cochlea directly stimulated by vibrations reaching it through the medium of the solid bones of the cranium, or is hearing by bone conduction merely a modified form of air conduction?

It is generally assumed that vibrating bodies of sufficient intensity and frequency, such as tuning forks, when brought in close contact with the cranial bones are capable of exciting the organ of Corti via the solid medium and thus produce the sensation of sound; yet there is no experimental evidence to substantiate this general belief.

Kerrison,[1] in discussing air conduction and bone conduction of sound, stated:

> Sound is brought to the organ of hearing in two ways, (1) by air conduction, as when the vibrating body is at a variable distance from the ear, the sound waves being transmitted through the medium of the surrounding air; and (2) by bone conduction, as when the vibrating body is in contact with the skull, the sound waves being propagated through the solid medium of the cranial bones.

Jackson and Coates,[2] in speaking of air and bone conduction, stated:

> Sound is conducted to the ears by air conduction, by bone conduction, or both. Air conduction is by way of the external auditory meatus, tympanic membrane, and ossicular chain to the internal ear; or it may go by way of the nose and pharynx through the eustachian tube to the middle ear and then to the inner ear. Bone conduction also has two pathways; either the craniotympanic, by way of the bones of the skull to the middle ear, and then to the inner ear; or craniolabyrinthine, through the cranial bones via the petrous portion of the temporal bone to the inner ear. According to Bezold the difference between air and bone conduction lies in the fact that by way of air the sound waves strike the flat surface of the drum membrane and ligamentum annulare, and by way of bone they impinge upon the edge thereof.

Politzer,[3] in treating of the same subject, expressed himself in the following terms:

> The vibrations of a body brought into contact with the cranial bones are distributed to all parts of the head, and thus reach the internal ear. These vibra-

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1. Kerrison, P. D.: Diseases of the Ear, Philadelphia, J. B. Lippincott Company, 1930.

2. Jackson, Chevalier, and Coates, G. M.: The Nose, Throat, and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929.

3. Politzer, Adam: A Textbook of the Diseases of the Ear, ed. 6, Philadelphia, Lea & Febiger, 1926.

tions reach the labyrinth in two ways: (1) by direct conduction through the solid bones to the labyrinth, and (2) by transmission from the cranial bones to the membrana tympani and ossicles, and from these to the labyrinth (craniotympanic conduction). Henson and Bezold doubt that the waves of sound are perceived by direct conduction through the cranial bones to the labyrinth, but assert that it takes place only through cranio-tympanic conduction. This view is contradicted by the fact that a still stronger perception through the cranial bones is observed when there is a complete loss of the drum membrane and ossicles.

As will be observed from the foregoing quotations, all the authors stress the importance of direct transmission of sound through the solid bones of the cranium to the internal ear. Kerrison, in fact, mentioned no other route of propagation, while the others named another way of sound transmission (craniotympanic). The latter avenue of sound propagation in hearing by bone conduction is relegated to a place of secondary importance in this process by the authors. Bezold, on the other hand, as indirectly quoted by Jackson and Coates and by Politzer, believed that the craniotympanic route is the only mode of sound transmission in bone conduction, and explained it on the basis that the sound waves strike the drum membrane and annulus tympanicus edgewise instead of impinging on its flat surface as is the case in air conduction, which explanation is not quite convincing.

Politzer, to prove that sound is directly transmissible through the skull and to refute Bezold's belief to the contrary, adduced the fact that when the tympanic membrane and the ossicles are completely destroyed a still stronger perception through the cranial bones is observed, and from this he logically concluded that the craniolabyrinthine route of sound propagation is the chief factor in hearing by bone conduction, while the craniotympanic route of sound conduction by bone is only auxiliary to the former.

Those who maintain that sounds by bone conduction are transmitted only through the medium of the tympanic structures (craniotympanic conduction) and those who uphold the view that they are transmitted through both the solid bones (craniolabyrinthine conduction) and the tympanic structures suffer alike in their inability to explain certain phenomena connected with hearing by bone conduction. This is due to the fact that both sides fail to take into consideration still another mode of sound transmission.

To quote the opening paragraph from one of my articles:[4]

When the bones of the skull are set in vibration by an activated tuning fork, or by any other body which is capable of imparting its vibrations to the cranium, these vibrations reach the labyrinth in three ways: 1, By the bones directly transmitting their vibration to the internal ear (cranial conduction); 2, by the

4. Becker, B. M.: Studies in Bone Conduction, Laryngoscope **40**:258 (April) 1930.

bones imparting their vibrations to the greater and lesser tympanic membranes and to the ossicular chain and through these carried into the labyrinth (craniotympanic conduction); and 3, by the bones imparting their vibrations to the air contained in the middle and outer ears, and this resonating air, in turn, acting upon the two tympanic membranes and chain of ossicles terminating in the stapedian footplate, and thus reaching the perceptive apparatus (craniopneumotympanic conduction).

It is my conviction that the third mode of sound propagation, as just described (craniopneumotympanic conduction), is the chief factor in hearing by bone conduction and that the craniotympanic route plays a subordinate rôle, as Politzer noted that when the tympanic structures are completely lost the sounds are perceived still better by bone conduction; no room is left for the belief that sounds reach the inner ear directly through the skull.

The proponents of the osteolabyrinthine route of bone conduction cannot satisfactorily explain some of the phenomena observed in connection with this mode of hearing, such as the intensification of sound in cases of conductive lesions of the middle ear and obstructive lesions in the external ear, the increase in the intensity of sound when a finger is introduced into the ear canal, the maximal increment of sound in the ear covered by the palm of the hand and the occurrence of cross or diagonal vibration.

The intensification and prolongation of sound in bone conduction as observed in tympanic lesions and when the ear is obstructed by a finger or covered by the palm of the hand are alike only in their results. They differ in their mode of causation and in their interpretation. While the explanation of the former is based on conjecture and hypothesis, the explanation of the latter rests on well known physical laws that can readily be demonstrated.

1. The air in all the skull cavities is agitated by the vibrating skull. 2. When a volume of air that is confined in a cavity is set in motion by a sound-producing object it responds with greater intensity than a similar volume of air that is unconfined. 3. The greater the size of the closed cavity, the more pronounced is the sound. This is precisely what is taking place when the ear is closed by the finger or palm and accounts for the increase in sound as a result of such closure.

EXPERIMENTAL DEMONSTRATIONS

(*A*) *Experiments Performed on a Dry Skull.*—1. Set a tuning fork in maximum vibration and hold it at a distance from the ear so that its sound cannot be heard. Now place its stem against the dry skull, and its sound becomes perceptible from a variable distance and for a variable length of time. 2. Repeat the foregoing instructions and place the ear against the vibrating skull, or apply the bell or the diaphragm of a

stethoscope to it, and the sound will become greatly intensified. I used a Fleischer stethoscope with a localizer (a small stem that is screwed in the center of the diaphragm by means of which small areas can be singled out for testing). 3. Set the vibrating fork against the teeth of the skull, and the sound is perceived stronger both from a distance and when the ear or instrument is applied to it. When the process is reversed, the fork is applied to any part of the skull and the localizer is applied to the teeth, the result is the same. This test, except for the definite increase of sound as heard from a distance on application of the fork to the teeth, is variable, depending on what forks are used and on the site of application of the ear or the stethoscope. Furthermore, it depends on the frequency of the fork whether the sound is heard from a distance, with the ear brought close to the skull, or whether the ear must come in contact with the skull to hear the sound. 4. When a vibrating fork is set on the skull, the sound, whether by ear or through an instrument, will be heard best at or near the site of application of the fork, and the next best place will be at a point opposite the zone of contact—cross or diagonal conduction. 5. When the ear is pressed against one temporal bone of the skull and a vibrating fork is placed against the other, the sound will be perceived louder and longer than when the fork is set against one's own mastoid at the side opposite that at which the ear is in contact with the bone. 6. Lastly, the labyrinthine capsule possesses no preeminence over the other bones of the skull in sound conduction, as was asserted by some supporters of the theory of a craniolabyrinthine route of bone conduction. This was tested both by application of the fork to the petrous portion of the mastoid and listening to the sounds at other parts of the skull and by application of the fork at any other part of the skull and listening to the sound, by means of the localizer, at the petrous pyramid.

(*B*) *Experiments Performed on the Skull of the Subject.*—1. Set a forcefully vibrating fork on the cranium of the subject and bring your ear close to his head and you will perceive a faint buzz emanating from the vibrating head. 2. Apply the ear to the patient's head or a stethoscope, and you will hear the sound better and longer than will the subject. 3. With the vibrating fork in contact with the mastoid of the subject, close his opposite ear by pressing the palm of your hand or his own against it and apply your ear over the temporal bone of the obstructed ear, and the sound of the fork will be perceptible by both for approximately the same length of time, a trifle longer by the subject. 4. Set the stem of the fork against the subject's teeth, and both you and the patient will hear the sound better. 5. Insert the ear tip of a rubber tube in your ear and hold the free end of it in either ear canal of the subject, taking care not to touch any part of the external ear. Now set a vibrating fork on the patient's skull, and the

sound will be distinctly heard. 6. Proceed as in the previous experiment, but instead of the free end of the tube lying in the open canal convert the latter into a closed cavity by applying your or the patient's open palm against it, and let the end of the tube pass between your or his fingers into the closed canal (care being taken to close all the space around the tube, and at the same time not to compress it), and the sound will become quite pronounced to you. If the fork is placed over one mastoid of the subject while the opposite ear is closed with the open hand and the examiner listens to the sound in the closed ear canal, both the patient and the examiner will hear the sound approximately the same length of time, the patient probably a trifle longer. This is so true that the examiner can inform the patient when he (the patient) stops hearing the sound by bone conduction.

(*C*) *Subjective Experiments on Oneself.*—1. Set the vibrating fork on the midskull, and when the sound is no longer heard close one of the ears with the palm, and the sound becomes perceptible again. 2. Place the fork over one of the mastoids, and the sound will be heard on the same side; now cover the opposite ear with the open hand, and the sound will immediately shift to the closed ear canal and will be heard from 50 to 100 per cent longer than with the open canal. 3. Place the ear tip of the tube in one canal and set a vibrating fork on the skull. When the sound is no longer perceived, place the free end of the tube in the mouth, pursing the lips around it, and the sound again becomes distinctly audible.

(*D*) *Correlation, Evaluation and Interpretation of Results Obtained From the Foregoing Experimental Demonstrations.*—The following propositions are either self-evident or are made evident by some of the experiments demonstrated: 1. The skull, whether dry or in the living state, acts as a sound box or resonator and is set in vibration when a sound-producing body of sufficient intensity and frequency is brought in contact with it, as is shown by demonstrations 1, 2 and 3 under *A* and by experiments 1 and 2 under *B*. 2. The contained air in all the cavities of the skull is set in motion by the vibrating skull and tends to reinforce the sound thus produced, as is shown by experiments 5 and 6 under *B* and experiment 3 under *C*. 3. The air in a closed cavity responds with greater force to a vibrating body than an equal volume of air in an open cavity, as becomes evident from experiments 3 and 6 under *B* and 1 and 2 under *C*. 4. The intensity and duration of perception of sound by bone conduction are in direct proportion to the intensity and duration of air disturbance in the external and middle ears caused by the resonating skull, as proved by experiments 3 and 6 under *B* and 1 and 2 under *C*. 5. Sound waves do not enter via the craniolabyrinthine route. If they did, the cochlea would remain unaffected by the greater agitation of the air in the obstructed ear, the

intensity of the vibration of the skull remaining the same. This is proved by experiments 5 and 6 under *B* and 1 and 2 under *C*.

The living skull responds to a vibrating body in contact with it as does the dry skull, but with less intensity owing to the absorption of some of the sound vibrations by the soft tissues investing it and by those contained in it. The process of hearing the fork sound by the examiner with his ear pressed against the patient's resonating skull is in all respects similar to that of the patient's hearing of it, and the difference, if any is present, is a question only of degree. This (the vibrating skull with the examiner's ear in contact with it) is a resonator with two delicately attuned perceptive apparatuses in contact with it: the one (the examiner's) placed on the outside of it, and the other (the patient's) placed on the inside. This latter circumstance accounts for the difference in the length of time the sound is heard by each. In both the resonating skull sets in motion the air in the external and middle ears, and from these cavities the sound vibrations are carried by the tympanic structures to the oval and round windows through which they are transmitted to the perilymph and endolymph and to the organ of Corti (craniopneumotympanic conduction). In the event of the loss of the tympanic structures, the air vibrations enter the labyrinth directly via the external and middle ears without the intervention of the drum and ossicular chain (craniopneumatic conduction).

SUMMARY

Sounds perceived by bone conduction enter the inner ear via the external ear and the middle ear with its component structures if these are intact, or they enter the internal ear directly through the two fenestrae if the drum and ossicles are destroyed.

CONCLUSION

Bone conduction is a modified form of air conduction.

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USE OF NEGATIVE PRESSURE IN OTOLARYNGOLOGY

REPORT OF A SERIOUS ACCIDENT

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Negative pressure today occupies a position in the armamentarium of the otolaryngologist. It is employed in the diagnosis and treatment of suppurative sinus diseases, as a means of therapy in otitic suppuration and in operations on the nose and throat under general anesthesia, and is indispensable in broncho-esophagoscopy.

The type of apparatus used at the present time is an electrically operated pump, with a rubber tube connecting the pump with a variety of applicator nozzles. The latter are made of glass, metal or hard rubber in various sizes suitable for application to the nostril. Interposed between the nozzles and the pump at some point is a bottle reservoir which collects the material aspirated and prevents the fluid from reaching the pump. The technic, briefly, is as follows: The applicator nozzle is applied to one nostril while the other is compressed by the patient's thumb. The patient is told to swallow or to say "K," which procedure brings the soft palate against the postpharyngeal wall and closes off the nasopharynx from the oropharynx. Negative pressure is applied during this time, the vacuum pressure gage attached to the apparatus being noted, or if this is not attached, the lowest possible pulling force being used, in order to avoid any harmful after-effect. Suction with the use of a nozzle is termed massive suction. A fine suction tip may be used in place of the applicator nozzle to provide finer suction. No name has been definitely given to this type of suction, but as the lumen of the tip is finer than the lumen of the nozzle, I shall term it capillary suction, though this is not strictly correct. At the bedside, where an electrically operated pump is not available, negative pressure can be employed by means of a hand type syringe with a bulb.

Bier,[1] in 1903, published a monograph entitled "Hyperemie als Heilmittel," which was destined to revolutionize therapy in the various branches of medicine and surgery. He emphasized the importance of

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From the Oto-Rhinologic Service of Dr. George M. Coates, Graduate Hospital of the University of Pennsylvania.

1. Bier, August: Hyperemia as a Therapeutic Agent, authorized translation by Gustavus M. Blech, Hammond, Ind., F. S. Betz Company, 1915, pp. 74 and 227.

hyperemia, stating that it is nature's method of reaction to any inflammation or irritation, the promotion of which is bound to be highly beneficial. He also constructed a series of suction apparatus incorporating Junod's principles, which were used particularly on the extremities and which produced hyperemia with reasonably good results.

Following Bier's monograph, Sondermann,[2] certain of the healing power of hyperemia, in 1905, commenced to apply suction to the nose. He found that he was able not only to "hyperemiate" the tissues but to effect physical cleanliness by removing secretions. His apparatus consisted of a mask for the nose, a ventilating tube and a suction ball. The mask was applied to the nose, the oropharynx closed off by the patient making a "K" sound and the closed suction ball permitted to expand. Sondermann felt that his apparatus helped somewhat in diagnosis, though it was difficult to recognize from which ostium the secretions drained. He obtained good results in a few cases of sinus suppuration and had no accident. Further researches convinced Sondermann[3] that if pus is recovered on suction and the same result is obtained on the following day it serves to strengthen the diagnosis.

Uffenorde,[4] in 1906, after studying a number of cases, concluded that while hyperemia was produced and some results obtained, suction did not give a really accurate diagnosis, nor could the sinuses be cleansed as thoroughly as by irrigation. Kyle[5] advised suction particularly when the eustachian tube had become involved in a catarrhal process.

Ballenger[6] stressed the value of negative pressure in acute, subacute and chronic infections of the upper respiratory tract, because it promotes reaction to inflammation, increases local nutrition and effects free drainage.

Brawley,[7] whose apparatus was attached to a water faucet, found negative pressure efficacious in his series of studies. He felt that suction

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2. Sondermann: Eine neue Methode zur Diagnose und Therapie der Nasenkrankungen, München. med. Wchnschr. **52**:17 (Jan. 3) 1905.

3. Sondermann: Weiterer Erfahrungen mit meinem Nasensauger, quoted by Skillern, R. H.: Accessory Sinuses of the Nose, ed. 4, Philadelphia, J. B. Lippincott Company, 1923, p. 85.

4. Uffenorde, W.: Kritische Bemerkungen über die Sondermannsche Saugmethode bei Erkrankungen der Nasennebenhöhlen, München. med. Wchnschr. **53**: 1165 (June 12) 1906.

5. Kyle, D. Braden: The Nose and Throat, Philadelphia, W. B. Saunders Company, 1899, p. 73.

6. Ballenger, W. L.: Diseases of the Nose, Throat and Ear, Philadelphia, Lea & Febiger, 1909, p. 46.

7. Brawley, Frank E.: Demonstration of the Suction Method in Diagnosing Sinus Disease, Laryngoscope **16**:530, 1906.

is a valuable method of conservative treatment, and that removal of decomposing secretions is equivalent to surgical drainage.

Negative pressure was studied carefully in fifty or sixty cases by Lewis,[8] who obtained such gratifying results in suppurative diseases of the ears and sinuses that he considered the method of first rank in the treatment of those conditions. He admitted, however, that it is not applicable to all cases, particularly those of hyperplastic sinusitis and chronic suppurative otitis media with cholesteatoma. His technic was as follows: Thorough shrinkage of the intranasal structures was obtained, followed by careful irrigation for the purpose of dissolving and thinning secretions. The head was then tilted in various positions so as to cause the contents of the sinus to overlie the natural opening or openings by gravitation, after which the negative pressure was applied as aforementioned. By these procedures he was able to provide freer drainage, better ventilation and hyperemia. For the nose and sinuses, any continuous negative pressure, such as Brawley's, or an electric pump was satisfactory, but for the ear he expressed a preference for Siegel's otoscope. Though Lewis expressed the belief that the induced hyperemia per se is adequate, by providing a higher opsonic content in the general circulation, he preferred to fortify the blood with vaccine therapy. He concluded that negative pressure affords the best results, because there are the least possible loss of tissue and the least disturbance of anatomic relations with the greatest possible conservation of function. Beck[9] highly commended the entire method, while Bryant[10] stated that he has been most successful since 1902 in treating selected patients with suppuration of the middle ear.

A few years later MacWhinnie[11] reported very good results in a series of cases of spheno-ethmoid disease by means of a vacuum apparatus that he constructed. The apparatus was used judiciously after careful shrinkage of the ethmoid structures. Following this he injected 50 per cent mild silver protein into the ethmoid cells by means of a very fine pipet, holding in all 2 cc.

In 1917, G. M. Coates,[12] reviewing the progress of otolaryngology, pointed out that the tendency was at that time toward conservative treatment of acute and chronic suppuration of the nasal accessory sinuses,

8. Lewis, E. R.: Negative Pressure as a Therapeutic Agent in Diseases of Nasal Accessory Sinuses, Throat, Ears and Mastoid, Tr. Am. Acad. Ophth., 1908, p. 346.

9. Beck, in discussion on Lewis (footnote 8).

10. Bryant, in discussion on Lewis (footnote 8).

11. MacWhinnie, A. M.: The Treatment of Infections of Accessory Sinuses, New York M. J. **103**:213, 1916.

12. Coates, George M.: Progress of Otolaryngology, Progressive Med., March, 1917, p. 263.

and that vacuum treatment had been used with some success for some time. He also described several types of apparatus: One, devised by Stillman, consists of a syringe operated by hand to produce suction and an ordinary compressed air nebulizer with a simple finger release. The second, by Harmon Smith, produces vacuum by means of a pump, after which the medicated solution is driven into the vacuumized cavities by the plunger action of a syringe. The third, by MacWhinnie, consists in the main of a pump and rubber tubing leading to a bottle reservoir. The fourth apparatus, by Coffin, introduces the principle of alternating positive and negative pressure and merits a brief description. It consists of two bottles mounted on the lower side of a tube ½ inch (1.27 cm.) in diameter called the connecting tube. The long end of the tube terminates in an olive pointed tip. Between the bottles is a switch key by means of which the inside of either bottle may be connected with the lumen of the connecting tube, the inside of the other bottle being at the same time disconnected. Between the bottles is a small tube extending through the key sleeve for the attachment of rubber tubing to the exhaust pump. When the key is so turned as to connect the second bottle with the connecting tube, one is able to create a vacuum by means of the pump. This will suck out any secretions from the sinuses on one side and form a partial vacuum. The olive tip is placed in one nostril, the other is closed tightly, and the patient by pronouncing the letter "K" closes the nasopharynx from the oropharynx. The first bottle is a nebulizer and is connected with the compressed air apparatus by the usual cut off. In it is medicated fluid. When the cavities have been sucked as dry as possible and a partial vacuum formed, the key is turned and the nebula forced in to replace the vacuum.

Coffin,[13] whose interest in negative pressure was first aroused by Haskin, made an intensive study in a series of cases of suppurative sinus disease. He obtained excellent results in children with the use of glass suction. He claimed no cure, but thought that the judicious use of negative pressure aids nature in the securing of a speedy return to health. He also stated that suction may conduce to hemorrhage, while in other cases this method is unsuitable. Following Coffin's interesting and lucid account of his experiences with negative pressure, a controversy took place on the merits of suction and the relative values of suction and irrigation in sinus disease. Coakley[14] stated that negative pressure will remove any secretions in the nose, but he could not see by what principle in physics negative pressure could be applied that would draw any substance, especially thin, watery fluid, out of the

13. Coffin, L. A.: Negative Pressure as a Therapeutic Measure in the Treatment of Sinus Disease, Laryngoscope **28**:882, 1918.

14. In discussion on Coffin (footnote 13).

inside of a cavity, viz., one with a rigid, bony wall. Further, the mucous membrane is apt to become swollen and edematous following negative pressure, which would actually interfere with free drainage from the various ostia. He concluded that suction is a valuable aid in the removal of secretions from the nose, but claimed better results from simple washing of the nose, and that irrigation is a much more thorough surgical procedure than suction for the removal of the inflammatory products within the sinuses. Dwyer [14] opposed this reasoning, claiming that the main value of suction is the hyperemia it produces, the chief aim being the production of an increased supply of blood. Having studied irrigation for some time, he was not highly impressed with the method of suction per se, though he admitted that irrigation and suction have their places, and that if the latter is properly used no harm can result. In conclusion he stated that the advantages of suction have been found to be so great that the Manhattan Eye, Ear and Throat Hospital has practically stopped the use of irrigation of the nose.

Hurd [14] emphasized the importance of suction in diagnosis, in operative procedures and as a means of cleaning out the nose, although it is of little value in therapeusis, according to his experience.

In concluding this discussion, Coffin stated that the principle of negative pressure is not to empty a sinus but to secure drainage, and, as is well known by the laws of physics, if one exhausts the air in the nostril, producing a low air pressure, the air in the sinus, the openings of which are closed, expands, thus driving out secretions. Negative pressure does not empty a sinus in the same way as washing it out, as Coakley stated. The chief aim is to secure free drainage and hyperemia. Coffin further stated that regardless of how thoroughly Coakley douched the nose to cleanse the sinuses, he could by suction take out as much or more pus as was removed by the douching, and that in his experience he has never had any accident or untoward result with the use of negative pressure, nor was drainage impeded or were polyps produced. Further experience with negative pressure convinced Coffin [15] that careful suction with the use of the various apparatus on the market not only drains the sinuses but produces a Bier's hyperemia of the lining membrane; this confirmed his observations of many years before.

Brühl, Politzer and Smith [16] were accustomed to follow free incision of the drumhead with aspiration, using Siegel's apparatus.

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15. Coffin, L. A., in Jackson, Chevalier, and Coates, G. M.: The Nose, Throat and Ear, Philadelphia, W. B. Saunders Company, 1929, p. 138.

16. Brühl, Politzer and Smith: Atlas and Epitome of Otology, Saunders Medical Hand Atlases, Philadelphia, W. B. Saunders Company, 1902, p. 186.

Smith [17] always taught the importance of applying suction following myringotomy with a view to relieving the congested mucosa of considerable blood and thoroughly evacuating the pent up secretions.

A. J. Wagers,[18] in 1921, published an excellent monograph on negative pressure, in which he advocated suction following myringotomy but cautioned against the use of too great pressure. He further stated that daily gentle suction should constitute part of the routine treatment of acute and chronic suppurative otitis media. On mastoids on which operation had been done suction could be performed with a curved tip medicine dropper. Sinus suction he performed in the following manner: The head was tilted in various directions so as to render the ostium or ostia dependent. A glass nozzle was applied to one nostril while the other nostril (the free one) was compressed by the operator's finger. Suction was applied in the usual way, but the free nostril was quickly released and compressed again, and this process was repeated until no more fluid was recovered. By releasing the free nostril, air was enabled to rush inward, backward and around the septum till it filled the cavities of the vacuumized nostril. Compression of the free nostril vacuumized the cavities of the occupied nostril.

R. H. Skillern [19] was at first impressed with the possibilities of negative pressure, but he encountered a number of difficulties that made him disappointed with this procedure. His failure to obtain good results with suction in diagnosis and treatment he attributed to the patient's inability to close off the choanae; but even if this were successful, the time of actual suction would be too short to draw much of the secretion out of the sinuses. In justice to the method, he felt that its greatest value was in the treatment of old ethmoid infections, but that it was for the most part unsatisfactory in frontal, maxillary and sphenoid infections. He expressed the belief, further, that reliable diagnostic conclusions in ethmoiditis could be obtained by any one skilled in the technic of negative pressure.

Tilley [20] was likewise disappointed with it, and in stressing free, spontaneous, permanent drainage he expressed a preference for irrigation,[21] claiming that it removes irritating products of bacterial activity and those incident to the inflammatory reaction of the mucosa.

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17. Smith, S. MacCuen: Common Diseases of the Ear, New York M. J. **103**:49 (Jan. 8) 1916.

18. Wagers, Arthur J.: The Use of Suction in Otolaryngology. Technique of Application, Therap. Gaz. **45**:80 (Feb. 15) 1921.

19. Skillern, Ross H.: The Catarrhal and Suppurative Diseases of the Accessory Sinuses of the Nose, ed. 4, Philadelphia, J. B. Lippincott Company, 1923, pp. 84 and 88.

20. Tilley, Herbert, quoted by Skillern (footnote 19, p. 85).

21. Tilley, Herbert: Some Experiences in the Surgical Treatment of Inflammation of the Frontal Sinus and Its Complications, Tr. Am. Acad. Ophth., 1929, p. 38.

St. Clair Thomson [22] favors the use of suction with a suitable apparatus in cases of sinusitis. Other authors, such as Logan Turner,[23] Hays [24] and Barnhill,[25] advocate suction.

Scal [26] recently reported a few cases in which deep-seated pathologic conditions of the mastoid resulted from suction applied to the ear following myringotomy. To such a condition he applied the term "suction mastoid." The mechanism of the suction mastoid as explained by Kopetzky [27] and Almour [28] is the production of negative pressure, which results in a backflow of pus into the aditus and antrum. The free flow of pus from the antrum is obstructed by the resultant engorgement and swelling of the mucous membrane and capillaries. Scal concluded that suction should never be applied to the ears.

Beck,[29] however, claimed excellent results with the use of skilful capillary suction in his cases of suppurating ears.

The removal of air from the sinuses in the displacement method of Proetz [30] is accomplished by negative pressure.

Faulkner [31] expressed a preference for a suction irrigator in cases of purulent discharge.

E. B. Gleason,[32] one of the oldest otolaryngologists from the point of view of service, and one of the outstanding ones in America, has employed negative pressure almost from its inception. He has never met with an accident as a result of using it. He has expressed the belief that suction should always be used judiciously and, when pain

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22. Thomson, St. Clair: The Nose and Throat, New York, D. Appleton and Company, 1928, p. 259.

23. Turner, A. Logan: Diseases of the Nose, Throat and Ear, New York, William Wood & Company, 1924, p. 66.

24. Hays, Harold: Diseases of the Ear, Nose and Throat, Philadelphia, F. A. Davis Company, 1925, p. 55.

25. Barnhill, John F.: The Nose, Throat and Ear, New York, D. Appleton and Company, 1928, pp. 132, 142 and 149.

26. Scal, J. Coleman: Suction Mastoids, Arch. Otolaryng. **9**:25 (March) 1929.

27. Kopetzky, S. J.: Otologic Surgery, New York, Paul B. Hoeber, Inc., 1925, p. 68; quoted by Scal (footnote 26).

28. Almour, Ralph: The Conservation of Hearing in the Suppurative Middle Ear Diseases of Childhood, M. J. & Rec. (supp.) **120**:178 (Dec. 17) 1924; quoted by Scal (footnote 26).

29. Beck, Joseph; Pollak, H. S., and Lederer, F.: Capillary Suction as an Aid in the Treatment of Ear, Nose and Throat Diseases, Laryngoscope **25**:402 (May) 1925.

30. Proetz, A. W.: Displacement Irrigation of Nasal Sinuses, Arch. Otolaryng. **4**:1 (July) 1926; The Displacement Method of Sinus Diagnosis and Treatment, St. Louis, Annals Publishing Company, 1931, p. 7.

31. Faulkner, E. Ross, in Jackson, Chevalier, and Coates, G. M.: The Nose, Throat and Ear, Philadelphia, W. B. Saunders Company, 1929, p. 86.

32. Gleason, Edward B.: Personal communication.

exists, with considerable caution, particularly in acute cases. His plan of thoroughly cleansing the sinuses is as follows: The intranasal structures are shrunk; gentle suction is then applied, after which a small amount of saline solution or compound solution of sodium borate, N. F., is instilled, and this in turn is followed by gentle suction. This process is repeated until the structures are thoroughly cleansed.

The bibliography on negative pressure is extensive, but I trust that the few references cited will suffice to indicate the trend of opinion regarding its efficiency.

That indiscriminate and injudicious suction can be harmful and produce serious consequences is well exemplified by the following case:

REPORT OF CASE

J. G., a woman, aged 27, was admitted to the private otorhinologic service of Dr. George M. Coates at the University of Pennsylvania Graduate Hospital on Dec. 21, 1931, with the following history, reported by Dr. J. D. Singleton, of that service. Her chronic complaint was of severe right frontal headache. She stated that the pain was constant, knifelike and sharp, and that she was unable to sleep and had to resort to the use of narcotics orally to alleviate slightly her terrific distress. History: The tonsils and adenoids had been removed fifteen years prior to admission; right middle turbinectomy and ethmoidectomy had been performed thirteen years before, left middle turbinectomy and ethmoidectomy eleven years before, submucous resection four years before, and appendectomy seven years before. The patient had had "sinus trouble" since tonsillectomy fifteen years previously, but the acute symptoms of severe right frontal headaches dated back two weeks previous to admission following a treatment at another hospital, at which time strong massive suction was applied. At that time "something seemed to turn over" in the patient's head. No bleeding occurred. A few hours later a severe headache developed which had persisted to the time of admission and was so severe as to require the use of strong sedatives.

General Examination.—The ears were normal. The cartilage of the septum of the nose had been removed, but there were thickening of the mucous membrane and a high deviation to the left. Both middle turbinates as well as a portion of the ethmoid cells had been removed. The mucosa of the nose was swollen and boggy, with secretions in both middle nasal passageways. There was a thick, glary discharge on the postpharyngeal wall. The tonsils were enucleated. There was a small tumor on the anterior border of the left sternocleidomastoid muscle, lying just beneath the skin, which had persisted since childhood. No abnormalities were noted in the chest or lungs. There was a scar at the appendical site. Otherwise the abdomen was normal. The extremities were normal. No abnormalities were found in the urine.

Roentgen Examination.—The frontal sinuses were asymmetrical; the left one was small and the one on the right was about five times as large and extended beyond the midline toward the left. There was considerable polypoid hyperplastic thickening of the mucous membrane lining with a supra-orbital extension of the right frontal sinus so that the right supra-orbital cell was almost continuous with the right frontal sinus. There was marked hyperplastic thickening of the mucous membrane lining of many of the cells of the ethmoid sinuses, especially the anterior cells, more on the right than on the left. On the left there was slight thickening of

the mucous membrane lining of the maxillary sinuses, and on the right marked polypoid hyperplastic thickening of the mucous membrane lining. There was slight thickening of the mucous membrane lining of the sphenoid sinuses on both sides. Roentgenograms taken in another hospital previous to admission showed a round mass, about 2 cm. in diameter, partially filling the right frontal sinus.

Operation.—A bilateral Skillern frontal sinus operation was performed on Dec. 22, 1931. The incision over the right brow was made in the usual manner. The periosteum was stripped. The cortex was found to be extremely thin; a bluish coloration about 2 cm. square was noted at the site of the right frontal sinus. When an opening was made in the sinus above the supra-orbital ridge and above the frontal articulation with the superior maxillary sinus about 2 fluidrachms of venous blood oozed out. A culture was made. The frontal sinus was large and contained abundant hyperplastic tissue, some of which was detached, and there were some polyps, particularly in the region of the right nasofrontal duct, giving an appearance as if herniation of polyps and mucous membrane had taken place therein. The hyperplastic tissue and polyps were removed, and the sinus was thoroughly cleansed of infected cells. The nasal process of the frontal bone was removed, and the right nasofrontal duct was cleansed of polyps, bone chips, etc. The incision was carried over to the left side. The cortex was healthy. A left supra-orbital ethmoid was noted, which was healthy and clean. This was not disturbed. One rubber drain was inserted in the right nostril for drainage. The anesthetic was avertin.

The patient made an uneventful recovery, and the course was afebrile. Staphylococcus albus was secured in pure culture. The patient was discharged from the hospital on Jan. 2, 1932.

COMMENT

Although it is the policy of this clinic to try conservative measures first in order to secure adequate drainage and ventilation in infections of the upper respiratory tract before resorting to operative treatment, it was decided, in view of the history and physical findings in this case, to undertake the investigation of the frontal sinus by the external route, for the reason that some hyperplastic tissue might have become detached from the underlying bone and by strong massive suction be compressed in the nasofrontal duct, giving rise to the symptoms mentioned. This was proved to be the case.

SUMMARY

1. Negative pressure has a certain value in the diagnosis and treatment of acute and chronic suppurative sinusitis.

2. It is valuable in the treatment of acute and chronic otorrhea.

3. Myringotomy should be followed with very gentle massive or capillary suction.

4. Negative pressure is indispensable in operations on the nose and throat, in laryngeal surgery and in endoscopy.

5. Negative pressure is valuable because it results in the least possible loss of tissue, the least disturbance of anatomic relations and the greatest possible conservation of function.

CONCLUSIONS

While it is true that suction has merits and uses, it is possible to use the method injudiciously, as was noted in the case report. To obviate such a possibility, the otolaryngologist should make it an immutable rule to employ negative pressure, either massive or capillary, with the utmost care, gentleness and skill.

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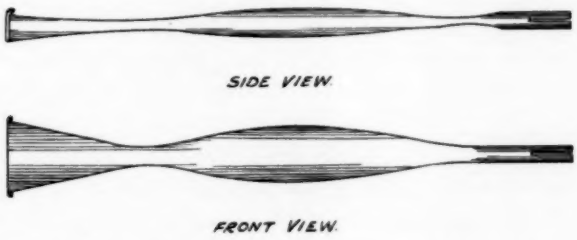
Clinical Notes

MODIFIED HURD TONSIL PILLAR RETRACTOR

LEE M. HURD, M.D., NEW YORK

The instrument presented is a modification of the familiar pillar retractor. The retractor end is approximately twice as wide as the old model, and is curved from side to side. The instrument is 9 inches (22.86 cm.) in length. At the handle end is a socket designed to admit the metal tip of the standard diathermy cord, for use when the retractor is employed as a dispersive electrode for electrocoagulation of the tonsil. When the retractor is being used for this purpose a piece of light rubber tubing is slipped over the handle end, until all but 1 inch (2.5 cm.) of the retractor end is covered, to prevent accidental burning of the lips, tongue or cheek.



Modified tonsil pillar retractor (one-third full size).

The retractor is of particular advantage in exposing the lower pole of the tonsil or the base of the tonsil fossa, as its width and curved surface permit of free retraction of the anterior tonsil pillar and, at the same time, good depression of the side of the base of the tongue. The instrument also allows retraction of the whole length of the anterior pillar for minute inspection of the upper part of the tonsil or its fossa when it is employed in the manner usual with the old model. The retractor is also useful in retracting the soft palate for inspection of the lower portion of the nasopharynx, as it offers a much better view than the old retractor because of its increased width and the curve of the blade.

39 East Fiftieth Street.

Presented before the American Laryngological, Rhinological and Otological Society, Atlantic City, N. J., May 23, 1932.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology for 1931

THE PARANASAL CAVITIES

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ANATOMY, PHYSIOLOGY AND EMBRYOLOGY

Hasty [1] lays particular stress on malnutrition in childhood as being responsible for developmental defects (pathologic anatomy) in the sphenoid and ethmoid cells. The malformation and variability make them easily susceptible to disease. The author makes the statement that disease in the ethmoid cells is probably the original focus of infection that subsequently involves other paranasal sinuses.

Williams [2] reported a case in which the patient complained of symptoms of maxillary sinus disease, and the roentgenogram revealed a few rudimentary cells occupying the normal site of the antrum.

Wenner [3] experimented by irrigating the antrums of rabbits with chemicals such as alizarin and oxalates. These substances have the property of precipitating the calcium ions normally found in the cells of the mucous membrane lining. After irrigations he found that the cilia became paralyzed, resulting in subsequent infection in the sinuses. He concludes, therefore, that calcium is necessary for normal ciliary movement in the antrum.

Anaphylactic reactions in the nose are accompanied by eosinophilia, a reaction that takes place in the capillaries or reticulo-endothelial system. If any means could be found to hinder or stop the reaction of eosinophilia, i. e., by endothelial blockage, it might also hinder or stop the anaphylactic reaction, according to Nemours and Wenner.[4] They experimented with nasal irrigations and intravenous injections of trypan

1. Hasty, E.: The Anatomy and Diseases of the Ethmoid and Sphenoid Sinuses, South. M. J. **24**:117, 1931.

2. Williams, C. B.: Rudimentary Left Antrum: Case Report, Texas State J. Med. **26**:763, 1931.

3. Wenner, W. F.: Effect of Calcium-Precipitating Substances on Ciliated Epithelium of the Maxillary Sinus, J. Lab. & Clin. Med. **16**:341, 1931.

4. Nemours, P. R., and Wenner, W. F.: Effect of Trypan Blue on Eosinophilia Experimentally Produced in Maxillary Sinus Mucosa, Ann. Otol., Rhin. & Laryng. **40**:852, 1931.

blue on rabbits in which eosinophilia had been previously produced with a calcium-combining substance such as alizarin. The mucous membrane of the rabbits was subsequently removed and examined for eosinophils. Their conclusions were that, first, trypan blue inhibits the production of experimental eosinophilia in the mucous membrane of the nasal sinuses by means of a blockade of the reticulo-endothelial system, and second, trypan blue has a modifying effect on the action of calcium-combining substances on the cilia of the mucous membrane of the maxillary sinuses.

Halle [5] discusses his findings in a case in which radical operation on the frontal sinus was performed, which seems to strengthen the theory that sinuses are necessary for the resonance and overtone of the voice. The patient's forehead protruded so extensively that he was often teased about the possible growth of horns. It was decided to remove the bony anterior wall, preserving the outer and inner periosteum in the hope that between the two a thin bony layer would later regenerate. After a small portion had been removed it was noted that the internal periosteum and mucous membrane of the frontal sinus vibrated synchronously with the respiratory movements. The pressure against the mucous membrane was more marked during deep breathing and when the patient talked than in superficial respiration. During the healing process it was found that when the finger was held against the region of the bone defect, a whirring, similar to pectoral fremitus, was perceptible. Its intensity fluctuated; it was more marked when the voice was raised and when the patient sang than when lower tones were produced. The author concludes that the large frontal sinus was an effective resonator which largely determined the quality of the voice. He suggests that roentgenographic studies of the sinuses of singers be made.

Childrey and Essex [6] experimented on dogs through a trephine opening into the frontal sinus. The natural ostium was sealed off with wax and drugs such as histamine, nicotine, ephedrine, rattlesnake venom and phenolsulphonphthalein were separately injected. Their effect on blood pressure and urinary output was compared to the effect of these same drugs injected subcutaneously into control animals, and was found to be negligible. They conclude that the margin of safety in the use of highly toxic drugs as therapeutic agents in the nose is very large, and that the absorption of bacterial toxins from infected sinuses is very slow. It is their opinion that sinusitis seldom acts as a focus of infection.

The intra-antral air pressure and the effects of respiration on antral drainage were studied by McMurray.[7] The author inserted a cannula

5. Halle, M.: Physiology of the Nasal Sinuses, Ztschr. f. Laryng., Rhin., Otol. **21**:46, 1931.

6. Childrey, J. H., and Essex, H. E.: Absorption from Mucosa of the Frontal Sinus, Proc. Staff Meet., Mayo Clin. **6**:345 (June 10) 1931.

7. McMurray, J.: Intra-Antral Air Pressure Incident to Respiratory Excursion: Effect on Antral Drainage, Arch. Otolaryng. **14**:581 (Nov.) 1931.

under the inferior turbinate and connected the end to a U-shaped tube which recorded the intra-antral pressure in millimeters of water. The quiet inspiratory reading was from —2 to —8 mm., and expiration from 0 to + 4 mm. Forced or prolonged respiratory excursions increased both the minus and the plus pressure. The ratio observed in ordinary respiration, however, remained the same. It is supposed that ordinary quiet respiration aids the drainage of the sinus by exerting a suction action during inspiration, the return of the pus into the antrum being prevented by a valvelike action of the hiatus semilunaris. An accessory ostium prevented an inspiratory negative pressure and allowed a greater expiratory positive pressure. Accessory ostia, therefore, might be a predisposing factor in maxillary sinusitis. An antrum with one opening not only drains better but protects itself against repeated infections. In order to prove the importance of the normal respiratory excursions through the nose on antral drainage, the author injected iodized oil into both antrums and sealed off one nostril with cotton and collodion. The unsealed side began emptying itself immediately. At the end of seventeen hours the sealed side was just as it was at the time of injection, as shown by a roentgenogram. The cotton was then removed, and the sealed antrum emptied itself in seven hours, thus proving the great importance of proper nasal breathing on antral drainage.

Grahe [8] experimented with rabbits in an effort to find some factors influencing the development of the nasal sinuses. Six young rabbits were deprived of their molar teeth on one side when they were 6 weeks old. Four months later the corresponding antrum was found to be undeveloped. In each of six other rabbits the external naris on one side was sewed up. This did not cause any abnormality of development in three cases. The inference is that the development of the maxillary antrums depends principally on the growth of the alveolus induced by mastication pressure, and that the development of the nasal sinuses is but little affected by the presence of nasal obstruction.

A demonstration of the embryologic development of the nasal accessory sinuses amply illustrated with drawings is contributed by Hanson.[9] He believes that the maxillary sinus primitively is an evagination of the mucous membrane of the floor or lateral wall of the infundibulum ethmoidale; that the frontal sinus develops either by a direct extension of the whole recessus frontales or from one or more of the anterior group of the ethmoidal cells or occasionally from the ventral extremity of the infundibulum ethmoidale; that the sphenoid sinus arises in relation to the posterior cupola of the cartilaginous nasal capsule and is merely a

8. Grahe, K.: Experimental Study of Development of the Nasal Sinuses, Acta oto-laryng. **15**:141, 1931.

9. Hanson, W. L.: Embryologic Development of Nasal Accessory Sinuses, Illinois M. J. **60**:386, 1931.

constricted portion of the nasal fossa, and that the ethmoidal cells are primarily evaginations from the middle, superior and supreme nasal meati.

Guggenheim [10] presents a preliminary report of a method of studying the paranasal sinuses, ear and osteogenesis of the bones of the head by taking roentgenograms of a number of embryos and comparing the progress of the parts in the different pictures.

Nemours [11] makes a comparison of the accessory nasal sinuses of man with those of the lower vertebrates. The primitive amphibia have no sinuses. Their nasal sinuses are ciliated or nonciliated according to whether most of their time is spent in the water. The first indication of a maxillary sinus is found in the higher amphibia, the epithelium being ciliated columnar. The adult alligator is the only reptile that has a maxillary sinus that corresponds somewhat to that of mammals. The maxillary sinuses of the horse, cat, dog, gorilla, orang-utan, opossum and kangaroo were compared.

Nemours states that the ethmoid sinuses of man have no homologous structures in the lower mammals. The sphenoid sinus does not exist as such in mammals below the primates. The corresponding cavity in the mammals is described in detail.

The frontal sinus in the lower mammals is more properly looked on as an anterior extension of the nasal cavity with the frontal bone to increase the olfactory area and cannot be regarded as a true sinus. It may or may not be filled with turbinal bodies.

From a phylogenetic point of view, the cause of pneumatization seems to be one of a lightening of the weight of the skull. Apparently the sense of smell has no influence on pneumatization. Sinuses of the various species must be compared by the location of the ostia and not on the extent of pneumatization.

Moriarty [12] points out clearly the exact anatomy of the anterior ethmoid artery within the ethmoid sinuses. It enters the ethmoid through the anterior ethmoidal foramen, passes horizontally through the ethmoid for an average distance of one-half inch (1.27 cm.) immediately below (pedal to) the base plate of the brain and then enters the cranial cavity through a slitlike opening at or very near its junction with the cribriform plate. It therefore makes an infallible landmark between the

10. Guggenheim, L. K.: Preliminary Report of Method of Studying Development of the Ear, Paranasal Sinuses and Various Centres of Ossification in the Head, Laryngoscope **41**:232, 1931.

11. Nemours, P. R.: A Comparison of the Accessory Nasal Sinuses of Man with Those of the Lower Vertebrates, Tr. Am. Laryng., Rhin. & Otol. Soc., Inc., 1931, p. 195.

12. Moriarty, R. W.: The Anatomical and Surgical Significance of the Anterior Ethmoidal Artery as Related to the Accessory Nasal Sinuses, Tr. Am. Laryng., Rhin. & Otol. Soc., Inc., 1931, p. 264.

brain and the ethmoid sinus, especially useful in external operations on the ethmoid sinus. The author cites instances in which the vessel had been injured during intranasal operation on this sinus.

PATHOLOGY AND BACTERIOLOGY

According to Shambaugh,[13] a definite basement membrane beneath the epithelium may be found anywhere in the respiratory tract. While it may be seen in normal appearing mucous membrane, it is probably evidence of a chronic epithelial irritation in the past. It usually occurs beneath pseudostratified columnar ciliated epithelium, but may occur with the transitional form and occasionally with stratified squamous epithelium. It is composed of a dense, more or less homogeneous layer of collagen beneath the epithelium and is interspersed with canaliculi, through which leukocytes migrate to the surface and perhaps tissue fluids escape to the surface. It is found in about a third of the specimens from chronic inflammations, and in about two thirds of the specimens from asthmatic patients. When greatly thickened, it is accompanied by a desquamating epithelium, often producing mucus excessively. This suggests that the presence of a basement membrane results from stimulation of connective tissue cells by abnormally active epithelium. Since edema, fibrosis and cellular infiltration of the lamina propria are not more marked when there is a thickened basement membrane, it has no clinical or pathologic significance other than being an evidence of chronic epithelial irritation, past or present.

Faulkner [14] describes the pathways by which a thrombus may extend to the cavernous sinus as follows:

1. Anterior foci with infection spreading through the angular, supraorbital and supratrochlear veins to the ophthalmic veins.

2. Internal foci through the ethmoidal veins or spreading through the walls of the sphenoid sinus.

3. Inferior foci with spread of infection through the pterygoid plexus or by direct proximal infection of the internal jugular through the lateral sinus and petrosals.

4. Posteriorly by extension from lateral sinus through the petrosals.

The anterior group of focal infections comprises such lesions as boils and carbuncles around the tip or ala nasi and infections of the eyelids. The internal group includes nasal sinus infections, such as acute exacerbation of chronic sinusitis and infections following intranasal operations. The inferior group embraces peritonsillar abscess, infection of the opera-

13. Shambaugh, G. E., Jr.: Basement Membrane in the Mucosa of the Upper Respiratory Passages, Arch. Otolaryng. **13**:556 (April) 1931.

14. Faulkner, E. R.: Rhinological Aspects of Cavernous Sinus Thrombosis, Surg., Gynec. & Obst. **52**:474, 1931.

tive field after tonsil operation, or after tooth extraction from the superior maxilla and deep cervical abscess.

The signs and symptoms of cavernous sinus thrombosis are well known: exophthalmos, swelling of the eyelids, chills with high temperature, fixed eyeball and death within a few days. The treatment varies according to the location of the primary foci. In the anterior group hot fomentations are advised. In the internal group pus should be evacuated, wherever found, by the Killian method. The author does not favor exenteration of the orbital contents. Ligation of the carotid on the affected side to put the part at rest has been tried, but its value is not yet proved. The transfusion of blood from immunized donors has been recommended by Eagleton.

Fenton [15] performed some experimental work on the frontal sinuses of cats in an effort to determine whether histiocytes could be increased in the mucous membrane after the injection of certain agents through a small opening in the anterior face of the sinus. Histiocytes are also called resting wandering cells, clasmatocytes or reticulo-endothelial cells and are the first line of defense or repair, appearing ahead of the polymorphonuclears and the lymphocytes. Repeated subcutaneous injections of trypan blue were used for selective staining of the histiocytes and the mucous membrane examined microscopically. Fenton concluded that histiocytes are normally present in small numbers near the blood vessels. Physical trauma and bacterial inoculation cause their arrival in considerable number. Rapid mobilization of polymorphonuclears seems to be favored by heat changes induced by hot paraffin. Solution of acacia, a typical colloid, destroys ciliary action. Human material removed surgically in cases of chronic sinusitis and placed in an incubator in contact with the dye revealed numerous histiocytes. Fenton's results suggest the devising of some method for artificially increasing the number of histiocytes in the sinus membrane and thus increasing the resistance and repair potentialities of these structures.

Hilding,[16] of the Mayo Clinic, discusses the influence of ciliary action on the bacteriology of the nose. He states that the protective mucous covering of the posterior two thirds of the nose is renewed once every six to ten minutes; that from the anterior third, every hour or two.

One hundred and six cultures were taken from the region of the middle turbinate of patients, two thirds of whom had "colds." Previous to this the nostrils had been closed with cotton pledgets for twenty

15. Fenton, R. A.: "Reticulo-Endothelial" Components of Accessory Sinus Mucosa: Experimental and Clinical Observations, Arch. Otolaryng. **14**:586 (Nov.) 1931.

16. Hilding, A.: Influence of Ciliary Activity on the Bacteriology of the Nose: Direction of Ciliary Currents on the Frontal Sinus of a Dog, Proc. Staff. Meet., Mayo Clin. **6**:320 (May 27) 1931.

minutes in order to allow the ciliated mucous membrane time to sweep itself free from saprophytes and contaminations derived from the air. Half the cultures were sterile; the other half revealed streptococci, staphylococci or diphtheroid bacilli. From these observations it may be inferred that the region around the middle meatus had had time to cleanse itself thoroughly of organisms in the twenty minutes the nose was plugged with cotton.

The direction and rate of drainage of the mucous secretion from the frontal sinus were studied in fifteen dogs at operation. Fine graphite particles were used to indicate direction and rate of drainage. The rate of flow was found to be entirely independent of gravity. The flow from the deepest portion of the cavity was directed upward, against the force of gravity. The rates of flow varied from 2 to 18 mm. a minute. Inactive areas were not found. The rate of flow accelerates as the ostium is approached.

Fetissof [17] advances two main theories to explain the pathogenesis of osteomas in the nasal sinuses: first, the theory of origin in remnants of embryonal cartilage, and second, that of their origin in the periosteum. It is thought that an osteoma may originate from remnants of cartilage remaining unossified in the ethmoid sinus and on the ethmoid bone. This is Arnold's view. The French school favors the second theory. The author describes an interesting case of osteoma of the right frontal sinus with recurrence after operation. He gives a considerable number of references to the literature on the subject.

A biochemical study of sinus disease in forty-eight patients was made by Israel and Nicholas.[18] In this study the removed bone, tissue contents of the sinus, pus and the patient's blood were subjected to chemical analysis for calcium, phosphorus, carbonate, chlorides, etc. The authors conclude by stating that they were unable to determine any characteristic or consistent chemical observations that were significant of bone, tissue or blood changes in sinus disease which could be looked on as having any bearing on its diagnosis, prognosis or treatment.

McGregor [19] presents a paper in which further proof of histologically normal regeneration of antral mucous membrane was claimed after it had been previously removed at operation. Twenty-two specimens from Dr. Mosher's laboratory at the Massachusetts Eye and Ear Infirmary and four from the University of Toronto form the material for the paper. "That the lining regenerates as normal mucous membrane over

17. Fetissof, A. G.: Pathogenesis of Osteomas of the Nasal Sinuses, Acta oto-laryng. **15**:85, 1931.

18. Israel, S., and Nicholas, H. O.: Biochemical Study of Diseases of the Nasal Sinuses, J. A. M. A. **97**:1453 (Nov. 14) 1931.

19. McGregor, G.: Further Proof of Regeneration of the Mucous Membrane in the Human Antrum, Arch. Otolaryng. **14**:309 (Sept.) 1931.

the whole of the antrum no one can say without examining every millimeter of the regenerated mucous membrane. In some places there is an increase of fibrous tissue; in some places it may be all scar tissue. However, the mucous membrane appears normal over the whole cavity and does regenerate." In the twenty-six specimens examined, all were ciliated except one. The outstanding observation was the presence in one case of new epithelium sixteen days after removal. Cilia had not had time to reform in this specimen. The author presents a detailed microscopic description of the method of repair and regeneration of mucous membrane. The paper is amply illustrated with photomicrographs.

Harris [20] presents four cases in which the Caldwell-Luc operation was performed and in which the microscopic changes in the bone and in the mucous membrane of the sinus could not be well correlated with the gross pathologic process seen at operation. Where the author expected to find many microscopic details to correspond with this pathologic process, he found little, and the converse was also true. He concludes with the hope that some day some genuinely good pathologist would turn rhinologist and correlate the gross and microscopic study of sinus disease.

Balmer [21] makes some interesting observations on the bacteriologic content of the maxillary sinus. With but few exceptions the literature teaches that the normal maxillary sinus, or antrum of Highmore, is sterile or free from bacteria. Torne, in 1903, demonstrated that healthy accessory sinuses of persons who had been dead over two hours were sterile. Lewis and Turner, in 1905, concluded after much investigation that the healthy accessory sinuses were probably sterile. Skillern, in 1912, examined post mortem the antrums of a hundred persons who died from tuberculosis, and he came to the conclusion that in health the sinuses were free from bacteria.

With a special technic so as to prevent contamination in obtaining specimens for bacterial examination from the antrum, Balmer compared a hundred normal and diseased antrums from a bacteriologic and clinical point of view. The antrum washings were examined scientifically. His investigations proved the fallacy of judging antral washing macroscopically. Turbidity is not a diagnostic proof of infection, since many were found to be sterile. Many clear washings were found to contain highly virile organisms. In fact, 76 per cent of the cases in the article in which the report was "washings returned clear" were found to be bacteriologically positive. Under normal circumstances the antrums are

20. Harris, R.: Gross and Microscopic Pathology of the Maxillary Sinus, South. M. J. **24**:662, 1931.

21. Balmer, F. B.: Relation of Clinical to Bacteriologic Observations in Normal and in Diseased Maxillary Antrum, Arch. Otolaryng. **14**:440 (Oct.) 1931.

free from bacteria. No obligate anaerobic bacteria were isolated. Staphylococci, streptococci, diphtheroid bacilli and pneumococci in the order named were the most commonly encountered organisms.

Turner and Loew [22] studied and compared the bacterial flora of the nasal cavity and middle ear of a series of rats before and after being deprived of vitamin A. Their conclusions were that lack of vitamin A was responsible for lowering the resistance of the mucosal lining of the sinuses toward invasion by bacteria. This was brought about by a change in the structure of the cell, which in turn changes the character of the protective mucous secretion.

Pugh [23] made a study of 101 known cases of sinus infection, making 135 cultures. Pure cultures were found as follows: pneumococcus, 8 times; Streptococcus haemolyticus, 8 times; Staphylococcus aureus, 7; Streptococcus viridans, 5, and nonhemolytic Staphylococcus aureus, 4 times. Mixed cultures were found in 98 instances in which Streptococcus viridans was found 39 times, the hemolytic and nonhemolytic streptococcus were each found 15 times; Staphylococcus albus 24 times, Staphylococcus aureus 19 times and pneumococcus 10 times.

Pugh also investigated the hydrogen ion concentration in the antrum and the relationship between the bacterial flora and the acid-alkaline reaction of the mucous fields. He concluded that no particular ion concentration either discouraged or hindered bacterial growth within the antrum. Another observation was that the bacterial flora of a diseased sinus changes continually.

BENIGN TUMORS

Shaheen [24] reports a case of psammoma in the maxillary antrum of a 12 year old Negro boy. The author considers its rarity as worthy of note. The Moure lateral rhinotomy incision was used to shell out the growth which completely filled the antrum and one nostril but which had no connection with the dura or ethmoid sinus. The origin of the growth was left undetermined.

Armitage [25] presents a case in which a frontal osteoma, measuring vertically 2½ inches (6.27 cm.) and horizontally 3½ (8.9 cm.) inches, was successfully removed and found to be attached by a small pedicle from the right infundibulum. The dura was slightly torn at operation, and was sutured.

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22. Turner, R. G., and Loew, E. R.: Infection of the Nasal Sinuses and Upper Respiratory Tract in Avitaminosis of Rats, J. Infect. Dis. **49**:244, 1931.

23. Pugh, J. W.: Nasal Sinus Infection, Laryngoscope **41**:697, 1931.

24. Shaheen, H. B.: Psammoma in the Maxillary Sinus, J. Laryng. & Otol. **46**:117 1931.

25. Armitage, G.: Successful Removal of an Osteoma from the Frontal Sinus, Brit. J. Surg. **19**:158, 1931.

The same author [26] also presents a historical survey of osteomas of the paranasal sinuses in association with a report of a large frontal osteoma, 6 by 6.5 by 7 cm., complicated by multiple mucoceles, one of which communicated with the lateral ventricle, causing a cerebrospinal rhinorrhea and a ventricular pneumatocele. The bony tumor was removed in two stages, and a third operation was necessary to close two cerebrospinal fistulas by means of fascial implants. The etiology of osteoma is obscure. Some authors try to explain it on an embryologic, traumatic or infectious basis. Osteomas of the frontal sinus form about 40 per cent of all osteomas of the sinuses. They usually occur in the second and third decades of life and more often in the male. They originate in the diploe of the frontal bone and are composed of hard ivory compact bone with scant haversian canals. They may grow by pressure atrophy in the direction of the eye, causing many ocular complications, or into the cranial cavity. Mucous secretion from any mucous membrane tissue included in the growth tends to collect into cystlike cavities called mucoceles which project through the dura mater into the brain substance.

Terracol and Lamarque [27] also describe a case of osteoma of the frontal sinus.

MALIGNANT TUMORS

The technic of treatment of early carcinoma of the antrum by irradiation is contributed in an exhaustive article by Sharp.[28] Early diagnosis is rarely made in this disease.

The symptoms and diagnosis are discussed. Roentgen examination is usually not of any help until after bone erosion. Reference is made to Martin and Ellis, who use a special technic of aspiration of the antrum to recover tissue for histologic study in early cases. Metastases are rare in any type of carcinoma of the antrum. The five types in the order of their frequency (Ewing) are: adenocarcinoma, schneiderian carcinoma, squamous cell carcinoma, basal cell and transitional cell carcinoma. It is the author's belief that infection plays an important rôle in the etiology.

Surgical exposure of the growth aims toward providing an accessible opening for implantation of radium. The growth is removed five days later. Accurate methods of calculation of radium and roentgen irradiation are given in the article to which the reader is referred. Irradiation is relied on rather than surgical measures. Involvement of

26. Armitage, G.: Osteoma of the Frontal Sinus, with Particular Reference to Its Intracranial Complications and with Report of a Case, Brit. J. Surg. **18**:565, 1931.

27. Terracol and Lamarque: Osteoma of the Frontal Sinus: Case Report, Gaz. méd. de France (supp. radiol.), no. 6, (Oct. 1) 1931, p. 133.

28. Sharp, G. S.: Technic of the Treatment of Cancer of the Antrum, Radiology **17**:984, 1931.

the floor of the orbit requires the exenteration of the orbit. Six representative case histories were given in detail.

Davison and Kahr [29] report two cases of carcinoma of the sphenoid sinuses with invasion of the cranial cavity. The neurologic signs were due to direct extension of the tumor into the middle and posterior fossae compressing practically all the cranial nerves. The sella turcica was invaded in one case and spared in the other. Meningeal signs were absent. No retrobulbar signs were evident. The authors state that in cases in which sinusitis is suspected that are not relieved by operation it is advisable to have repeated roentgenograms taken of the sella and posterior sinuses.

In answer to a statement that roentgenographic examination usually helped only in the late cases, Habbe [30] reports a case of epithelioma of the ethmoid sinus in which a definite roentgenographic diagnosis was made seven weeks prior to positive clinical signs of tumor.

Ohngren [31] gives a short account of the principles to be observed in remedying by plastic operations the deformities and defects in the face and palate after operation by diathermy on malignant disease of the nose and sinuses. For the larger defects, he finds the tube flap method of great value. Brief notes of five cases illustrated by photographs are given to lay stress on important points.

FRONTAL SINUS

Ersner and Pressman [32] describe their method of securing nerve block anesthesia for the radical operation on the frontal sinus. Procaine hydrochloride, 2 per cent, is injected into the supra-orbital nerve and the lateral wall of the middle meatus (or cocaine on cotton pledget for the latter). The authors claim complete and satisfactory anesthesia from these two injections. An optional injection is along the site of incision. The patient is prepared with morphine and scopolamine.

Sagols [33] discusses the treatment of acute frontal sinusitis. It is generally agreed that no surgical operation should be performed in an acute infection unless urgent. He advises control of pain by drugs and recommends the usual nasal shrinking treatments. The author considers that in cases of severe pain the internal nasal and external frontal nerves

29. Davison, C., and Kahr, S.: Cancer of the Sphenoid Sinus with Invasion of the Cranial Cavity, Arch. Otolaryng. **14**:16 (July) 1931.

30. Habbe, J. E.: Cancer of the Ethmoid Sinus, Radiology **16**:548, 1931.

31. Ohngren, G.: Plastic Surgery of the Face Following Electrocoagulation Treatment of Cancer of the Nasal Sinus, Acta oto-laryng. **16**:292, 1931.

32. Ersner, M. S., and Pressman, J. J.: Radical Operation on the Frontal Sinus Under Local Anesthesia, Arch. Otolaryng. **14**:26 (July) 1931.

33. Sagols, H.: Methods of Treatment in Acute Sinusitis, Ann. d'oto-laryng., 1931, p. 754.

should be blocked, and he describes the various methods for carrying this out. The indications for operation by the endonasal and the external routes are discussed and the operative technic described. The author also discusses in detail the surgical treatment of periorbital suppurations in which he does not appear to deal with the infected ethmoidal cells or to attempt drainage through the nose.

Howarth [34] discusses the treatment of chronic frontal sinusitis. Mechanical factors in the nose predisposing toward chronicity were enumerated. Small intranasal operations were tried first. Intranasal frontal and ethmoid exenteration after Mosher were next tried, followed by the radical external operation if unsuccessful. The author firmly believes that frontal sinus disease is always associated with disease of the ethmoid sinus and, therefore, both sinuses must be drained simultaneously. The Howarth operation consists of complete removal of the floor of the frontal sinus, the os planum of the ethmoid sinus and all the ethmoid cells up to the base of the skull. In some cases the author used skin grafting in order to keep the nasal opening intact.

Harmer and Russell [35] describe the routine operation carried out in sixty-three cases of frontal sinusitis at St. Bartholomew's Hospital when intranasal treatments are unsuccessful. It consists in making a small incision at the medial third of the eyebrow, opening into the frontal sinus, and passing a soft rubber tube over a properly fitting wire through the infundibulum into the nose. The tube extends through the external opening. The aim of the tube is to dilate the bony frontonasal canal by elastic pressure, without damaging its lining membrane. The authors are extremely careful, therefore, that the rubber tube is the proper size at insertion. As absorption of the bone takes place, the tube is replaced by a larger one. Irrigations keep the tube and sinus clean. In some cases it is necessary to bite away bone to make a canal for the tube into the nose. This is discouraged since lack of trauma is the keynote of success in this operation. The authors claim for this procedure simplicity, speed, no disfigurement and successful results.

Fraser and Stewart [36] present a thorough statistical investigation of frontal sinusitis encountered in their ward and private cases over a period of twenty-four years. They report on 241 acute and 97 chronic cases (over six months).

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34. Howarth, W.: Treatment of Chronic Frontal Sinusitis, Proc. Roy. Soc. Med. (Sect. Laryng.) **24**:34, 1931; J. Laryng. & Otol. **46**:397, 1931.

35. Harmer, D., and Russell, B.: Treatment of Frontal Sinusitis by Intubation: Analysis of Sixty-Three Cases, J. Laryng. & Otol. **46**:384, 1931; Proc. Roy. Soc. Med. (Sect. Laryng.) **24**:31, 1931.

36. Fraser, J. S., and Stewart, J. P.: Frontal Sinusitis: Statistical Investigation, Proc. Roy. Soc. Med. (Sect. Laryng.) **24**:23, 1931; J. Laryng. & Otol. **46**: 369, 1931.

Of the 241 persons with acute cases, 161 were treated conservatively by intranasal treatments or small local intranasal operations. Fourteen were operated on by the external route, on 11 of whom the obliterating operation was done (anterior wall and floor removed), 1 by the Killian and 2 by the modified Howarth (Lynch) procedures. Four died (3 after the obliterating operation), 5 were cured, 1 was improved and 4 were not heard from.

Of the 97 persons with chronic frontal sinusitis, 40 were operated on by the external route, on 9 of whom the modified Howarth procedure was used, on 2 the Killian procedure, and on 29 the radical obliterating operation. Results in the chronic cases were: 21 patients were cured, 2 improved, 14 were not heard from and 3 died.

The authors admit a considerable amount of deformity after the obliterating operation but claim a greater margin of safety in this operation over the Killian method. In acute cases, in the future, they intend to use the Howarth rather than the obliterating operation. They prefer the incision through the unshaven eyebrow rather than beneath it. They consider that the erect position causes the periodicity of pain in frontal sinus disease. Roentgenographic diagnosis of frontal sinusitis cannot always be relied on. The authors agree that the obliterating operation had been done too often in acute cases.

Uffenorde [37] discusses the radical treatment of suppuration of the frontal sinus in which he stresses avoiding deformity and permanent drainage into the nose. The author considers the Killian operation lacking in its cosmetic results and, therefore, does not remove the anterior wall except in rare instances of trauma or bone disease. He favors the Jansen-Ritter operation in which the floor of the frontal sinus, the ethmoidal cells, lacrimal cells, nasal bone and frontal process of the superior maxilla are removed and mucoperiosteal flaps formed to line the wide frontonasal passage. The lining of the frontal sinus is removed when necessary by curets with a malleable shaft.

Svenson [38] discusses his series of seventy-two external operations on the frontal sinus by the Killian method. He describes the technic of anesthesia and operation, the changes in the bone and mucous membrane at operation, the course of recovery, relapses and his results. He outlines the history of the external operation on the frontal sinus from the earliest time down to the Killian technic. The author is convinced that in his series with one exception the frontal sinus was filled with granulations after Killian's operation. In the lateral and upper portions these tissues were more dense than in the other parts. He considers

37. Uffenorde, W.: Radical Treatment of Suppurative Frontal Sinusitis, Acta oto-laryng. **16**:117, 1931.

38. Svenson, L. S.: Results of the Killian Operation on the Frontal Sinus, Monatschr. f. Ohrenh. **65**:557 and 664, 1931.

postoperative diplopia due to misplacement of the orbital fascia, and is careful to approximate the periosteum to its original position. He discusses also the cosmetic results and postoperative neuralgias.

Lillie [39] enumerates the postoperative complications in 158 consecutive case of radical external operation on the frontal sinus. The complications encountered numbered 45. In his opinion there seems to be no way of determining beforehand what complication, if any, may follow operation. He advocates the intratracheal use of ether, use of less catgut at operation, gentleness in handling tissue and avoidance of severe traction. The complications encountered in his series were: diplopia (transient), swelling of the upper eyelid (9 cases), orbital cellulitis with subsequent orbital abscess or corneal ulcer (treatment by moist heat), hematoma of the lid, chemosis of the conjunctiva, localized osteomyelitis with sequestration at the orbital ridge or floor of the sinus (5 cases), subperiosteal abscess, tonsillitis, erysipelas (4 cases), lung abscess (aspiration, 1 case), 2 cases of meningitis and a case of frontal lobe abscess. The patients in the last 3 cases died. No complication of generalized osteomyelitis was observed in this series of 159 radical operations.

Halle [40] recommends conservative treatment in acute suppurations of the accessory sinuses. However, in cases in which all conservative treatment had failed and in which symptoms were so severe as to need intervention, he agrees to intranasal opening of the frontal sinus and, if need be, a submucous resection of the septum for better drainage.

Fruhwald [41] of Vienna, in a paper on frontal sinus disease, discusses etiology, bacteriology, pathology, signs, symptoms and nonsurgical and surgical treatments in acute and chronic cases. A description of the Halle intranasal operation on the frontal sinus together with the author's modification is given in detail. The external operation is not discussed.

Burgess [42] reports a fatal case of frontal sinusitis in which operation was done by the external route; autopsy disclosed frontal lobe abscess, extradural abscess, osteomyelitis of the frontal bone and necrosis of the roof of the orbit. The striking feature of the case was the variation of the type and origin of the infecting organism at different periods of the illness. In the early stages, Staphylococcus aureus was predominant, the source being the frontal sinus. Later a hemolytic streptococcus septice-

39. Lillie, H. I.: Postoperative Complications of Radical External Operation on the Frontal Sinus, Arch. Otolaryng. **14**:793 (Dec.) 1931.

40. Halle, M.: Endocranial Complications in Nasal Sinus Diseases: Operations on the Septum, Monatschr. f. Ohrenh. **65**:425, 1931.

41. Fruhwald, V.: Inflammation of the Frontal Sinus: Its Conservative and Endonasal Operative Treatment, Laryngoscope **41**:81, 1931.

42. Burgess, J. A.: Frontal Sinusitis with Intracranial Suppuration and Invasion of the Blood and Cerebro-Spinal Fluid by Different Organisms, Brit. M. J. **1**:936, 1931.

mia developed, its origin being from the patient's lung. Finally, Pneumococcus mucosus and Staphylococcus aureus were found in the brain abscess, while type III pneumococcus was responsible for the terminal cerebrospinal meningitis.

Buckingham [43] reports a case of fulminating frontal sinusitis and orbital cellulitis which produced proptosis of the eye with no movement. At operation no pus was found, but the lining of the frontal sinus was polypoid. The moribund patient made a prompt recovery.

Triossi [44] reviews a case of giant mucocele of the frontal sinus. Of the classic triad of clinical signs, a slowly progressive tumor at the inner angle of the orbit and exophthalmos downward and temporally with diplopia associated with a lessened acuity of vision were present. The third sign, a naso-orbital hyperostosis at the junction of the frontal, nasal, ethmoid and maxillary bones causing a characteristic change at the root of the nose and of the facial expression, was absent.

The unusual secondary features were proptosis, a nonpalpable dehiscence of the floor of the sinus and negative results of rhinologic examination and exploratory puncture. Roentgenographic examination showed typical bone absorption by pressure. Radical operation through the eyebrow revealed 25 cc. of a chocolate-colored mucoserous fluid containing large numbers of typical cholesterol crystals.

Rosenberger and Kinney [45] report a mucocele of the frontal sinus in a 13 year old boy, which they consider interesting on account of the patient's youth.

Seiberling [46] reports a case of fracture of the mandible, superior maxilla, ethmoid sinus, frontal sinus and cribriform plate into the dura with escape of cerebrospinal fluid. Autogenous vaccine was instilled into the nose after the Proetz displacement method. The patient recovered.

Westervelt [47] describes the osteoplastic flap modification he uses for the radical operation on the frontal sinus instead of the obliterative procedure. This consists of an incision through the eyebrow and a vertical incision upward from the medial border. The skin is elevated upward. By making a series of openings through the anterior face of the sinus with a burr and using the Gigli saw to connect them, the

43. Buckingham, R. E.: Fulminating Frontal Sinusitis and Orbital Cellulitis, M. J. Australia **1**:201, 1931.

44. Triossi, S.: A Case of Giant Mucocele of the Frontal Sinus, Riv. otoneuro-oftal. **8**:63 (Jan.-Feb.) 1931.

45. Rosenberger, H. C., and Kinney, C. E.: Mucocele in the Frontal Sinus: Occurrence in a Boy Aged 13, Arch. Otolaryng. **13**:597 (April) 1931.

46. Seiberling, J. D.: Fracture of the Frontal Sinus and Ethmoid Through the Dura, Orbit and Maxillary Sinus: Case Report; A Phenomenal Recovery, Laryngoscope **41**:93, 1931.

47. Westervelt, H. O.: Radical Operations on the Frontal Sinus, J. Michigan M. Soc. **30**:848, 1931.

anterior bony wall with its adherent periosteum is laid downward in one piece over the eye as an osteoplastic flap, after which the frontal lining is cleaned out and a large opening made into the nose. The floor of the frontal sinus is also removed. The flap is then replaced, a deformity thus being prevented.

Kegel [48] discusses an unusual case of mucocele of the frontal sinus.

Other contributions on some phase of frontal sinus disease have been made by van der Hoeven,[49] Bisi,[50] Tesone,[51] Nasiell,[52] Patel,[53] Söderberg,[54] Fischer,[55] Stupka,[56] Ill,[57] Voss,[58] Alloway [59] and Gaskins.[60]

THE ETHMOID SINUS

From a series of a hundred external ethmofrontal operations, Simpson and Sanders [61] came to the following conclusions:

1. Morphine and scopolamine (three preoperative doses) combined with local anesthesia has advantages over general anesthesia.

2. A short incision is all that is necessary.

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48. Kegel, R. F. C.: Unusual Cyst of the Frontal Sinus-Mucocele, China M. J. **45**:449, 1931.

49. van der Hoeven, Leonard J.: New Files for Intranasal Operation in Sinusitis, Ann. d'oto-laryng., 1931, p. 295.

50. Bisi, H.: Frontal Sinusitis with a Fistula of the Upper Eyelid; Bilateral Maxillary Sinusitis with Osteitis of the Right Side; Cure with Operation: Case Report, Rev. de especialid. **6**:826, 1931.

51. Tesone, P.: Breaking of a Curette During an Operation for Frontal Sinusitis, Rev. de especialid. **6**:197, 1931.

52. Nasiell, V.: Therapy of Empyema of the Frontal Sinus by Lothrop-Sebileau Method: Presentation of a Successful Case, Acta oto-laryng. **16**:552, 1931.

53. Patel: Fracture Due to Depression of the Anterior Wall of the Frontal Sinus, Lyon chir. **28**:131, 1931.

54. Söderberg, F.: Results of the Bárány External Operation of the Frontal Sinuses, Acta oto-laryng. **16**:231, 1931.

55. Fischer, H.: Surgery of the Frontal Sinus by the Endonasal Route, Bull. et mém. Soc. d. chirurgiens de Paris **25**:95 (Feb. 20) 1931.

56. Stupka, W.: Plastic Methods for Correcting a Depression of the Frontal Sinus, After External Operations: Celluloid Plates, Monatschr. f. Ohrenh. **65**:39, 1931.

57. Ill: Serous Meningitis During Acute Frontal Sinusitis, Presse méd. **39**:78, 1931.

58. Voss, O.: Operative Cure of a Gunshot Wound Passing Through the Frontal Lobe with Involvement of the Frontal Sinuses, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **129**:69 (June 6) 1931.

59. Alloway, F. L.: Frontal Sinus Infection Followed by Osteomyelitis of the Skull: Case Report, M. Bull., Vet. Admin. **7**:1077, 1931.

60. Gaskins, D. R.: Cerebral Embolus Following the Lynch Radical Frontal Sinus Operation: Case Report, with Recovery, Southwestern Med. **15**:331, 1931.

61. Simpson, W. L., and Sanders, S. H.: Interesting Observations in More Than 100 External Ethmo-Frontal Operations, Laryngoscope **41**:23, 1931.

3. There is very little bleeding, which can be controlled without ligatures.

4. By the use of mirrors the entire lining mucosa of the sinus can be removed.

5. Good results have been obtained where the outer bony angle of the frontal floor has not been removed. (One wonders whether the good result will be permanent.)

6. The middle turbinate is retained—most of the lamina papyracea is retained.

7. Subcutaneous sutures are unnecessary.

Peters [62] presents a paper dealing with conservative intranasal operation on the sinuses. While he considers his operation safe, he gives a warning note that fatalities may follow such a simple procedure as removal of the anterior tip of middle turbinate. Nothing new is given in the paper.

Risch [63] reports a case of a submucous resection of the septum followed by tonsillitis, mastoiditis and meningitis, presumably as a complication from the ear. However, at autopsy the cause of the meningitis was seen to be in the ethmoid sinus, probably an acute exacerbation of a "closed ethmoiditis." He quotes Hajek as classifying ethmoiditis in the ratio of one open to two closed. In open empyema of the ethmoid sinus pus flows into the nasal cavity, whereas in closed empyema it is blocked and by pressure on the walls soon leads to dilatation or periostitic changes of the bony wall. As a rule, closed ethmoiditis manifests itself in a bullous formed middle turbinate or the bulla ethmoidalis. At times no pathologic process can be found in the nose. Risch cites instances in which a simple procedure such as removal of the anterior tip of the middle turbinate was followed by meningitis due to closed ethmoiditis. He makes a plea for routine autopsies in fatal cases.

Tremble [64] reports a case of a large mucocele of the ethmoid sinus which was opened intranasally, and in which iodized oil was subsequently injected in order to show its extent.

Ethmoid sinusitis is also discussed by Dufourmentel,[65] Leroux [66] and Duerto.[67]

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62. Peters, E. A.: A Safe Method of Dealing with Ethmoiditis, Brit. M. J. **1**:56, 1931.

63. Risch, O. C.: Meningitis Dependent upon Closed Ethmoiditis: Report of a Case with a Plea for Routine Autopsy Proctocols, Ann. Otol., Rhin. & Laryng. **40**:508, 1931.

64. Tremble, G. E.: Method of Outlining the Mucocele of the Ethmoid Sinus by Injection of Iodized Oil, Arch. Otolaryng. **13**:836 (June) 1931.

65. Dufourmentel, L.: Differential Diagnosis and Treatment of Diseases of the Ethmoid Sinus, Prat. méd. franç. **12**:91, 1931.

66. Leroux, L.: Clinical Aspects in Diseases of the Ethmoid Sinus, J. de méd. de Paris **51**:386 (May 7) 1931.

67. Duerto, J.: Retro-Orbital Abscess of Fronto-Ethmoidal Origin: Case Report, Arch. de med., cir. y especialid. **34**:1005 (Oct. 31) 1931.

Toti [68] presents a case of mucocele of the ethmoid sinus with a fistula into the lower eyelid. Ts'en [69] and Wong [70] present similar cases.

O'Brien [71] reports a case of meningocele within a middle turbinate which had been removed together with nasal polypi for the relief of nasal discharge. Clear cerebrospinal fluid was immediately seen to escape. The following day the patient showed signs of a localized meningitis with elevated temperature, headache, drowsiness and paralysis of the sixth, seventh, eighth and ninth cranial nerves. The patient eventually recovered. It was thought that the meningocele was present at birth, resulting from a congenital absence of the roof of the ethmoid sinus which allowed the dura to descend.

THE MAXILLARY SINUS

The rôle of latent maxillary sinusitis in the production of referred pains to other parts of the body is discussed by Paterson [72] and illustrated with x-ray films and case histories. A latent maxillary sinus is described as one producing secondary complications which dominate the picture, while the symptoms referable to the primary focus in the antrum are either absent or surprisingly slight. Referred symptoms causing ocular disorders such as retrobulbar neuritis, cerebral symptoms with marked headache simulating encephalitis or brain tumor, pain to different parts of the face and head (tic douloureux) and pulmonary complications such as bronchiectasis or asthma are described in case histories. Often no pus was seen in the nose. The diagnosis was made by roentgenograms and antrum puncture.

Russell [73] contributes a paper dealing with the importance of the nose in the production of faucial and pharyngeal complaints in patients who have a sore throat after clean tonsillectomy. The author contends that pus from the nose, especially the antrum, serves as the etiologic factor.

The physiology of the nose and throat is reviewed, attention being called to the ciliary action in the nose. Clinical features of this so-called "nasal sore throat," such as an onset with a cold and repetition with each cold, asymmetry of symptoms and signs in the throat, finding of

68. Toti, E.: Ethmoidal Mucocele with a Fistula into the Lower Eyelid: Case Report, Valsalva **7**:438, 1931.

69. Ts'en, S.: A Case of Mucocele of the Ethmoid Sinus, China M. J. **45**:453, 1931.

70. Wong, A. S.: Mucocele of the Left Frontal Sinus and Ethmoidal Cells, China M. J. **45**:991, 1931.

71. O'Brien, G. R.: A Meningocele Within the Middle Turbinate Discovered During an Operation on the Ethmoid Resulting in Meningitis and Recovery, Arch. Otolaryng. **14**:399 (Sept.) 1931.

72. Paterson, R.: Latent Maxillary Sinusitis, Lancet **2**:117 (July 18) 1931.

73. Russell, B.: Sore Throats of Other Than Tonsillar Origin, Brit. M. J. **2**:485, 1931.

mucopus by posterior rhinoscopy or beginning discoloration and thickness of the mucous membrane over the middle turbinate associated with a sore throat, should call attention to its probable etiology in the antrum or posterior ethmoid sinus. The author relies strongly on transillumination and makes a plea for early intranasal antral operation before gross changes in the antrum have irreparably damaged the ciliary action.

Koch [74] discusses the operative treatments of acute and chronic suppurations of the maxillary sinus. He evaluates the various operative methods in vogue and stresses the disadvantage of injuries to the teeth in the radical procedures. He favors an endonasal opening which he obtains by a drill and punch followed by irrigations. In hyperplastic and polypous changes of the mucous membrane, the Caldwell-Luc operation is indicated.

Benjamins and Huizinga [75] make an evaluation of the various surgical methods employed in the treatment of maxillary sinusitis on 250 patients. They found that the Caldwell-Luc operation resulted in cure in 80 per cent of the cases, whereas the endonasal operation effected cure in 60 per cent. Nevertheless, they favor the latter in many cases on account of its simplicity. Radical operation is necessary in cases in which polypoid changes exist in the mucous membrane. The authors also give a survey of the disorders that may follow after the operation, special attention being given to disturbances of the lacrimal canal and of the teeth.

A description in detail of the Caldwell-Luc operation under local anesthesia is presented by Voorhees.[76] He feels that the soothing words of the surgeon preoperatively do more good than a sedative. Procaine hydrochloride, 0.5 per cent, is injected into the region of the primary incision, the infra-orbital, the anterior and the posterior palatine nerves. The description of the operation itself follows in detail. The author feels that the operation under local anesthesia is so simple and easy that one should not temporize in patients who do not respond quickly to minor intranasal surgical measures or medical treatment.

Reese [77] describes a treatment of chronic maxillary sinusitis by zinc ionization and insufflation of iodine boric powder and states that with the exception of carcinoma, osteomyelitis or syphilis, all chronic inflammatory conditions of the antrums are curable by this method of treat-

74. Koch, J.: Operative Treatment of Acute and Chronic Suppuration of the Antrum, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **128**:16 (May 11) 1931.

75. Benjamins, C. E., and Huizinga, E.: Operative Treatment of Chronic Maxillary Sinusitis, Monatschr. f. Ohrenh. **65**:316, 1931.

76. Voorhees, I. W.: Radical Nasal Antrotomy Under Local Anesthesia, M. Times & Long Island M. J. **59**:105, 1931.

77. Reese, F. L.: Treatment of Chronic Sinusitis by Zinc Ionization and Iodine Boric Powder Insufflation, Ann. Otol., Rhin. & Laryng. **40**:264, 1931.

ment. Preparatory to the zinc ionization, the author makes a window in the naso-antral wall under the inferior turbinate through which he introduces his instruments of ionization. Doubt arises whether the cure comes as a result of the window, the ionization, the insufflations of iodine boric powder or the beneficial warm dry weather of Arizona where the treatments were carried out.

Ersner, Pressman and Ersner [78] offer a definite systematic technic of block anesthesia for the Caldwell-Luc operation based on the neuroanatomy of the antrum. Their injections of 2 per cent procaine hydrochloride are preceded by the administration of scopolamine and morphine. Their technic for anesthesia, which includes five injections, is as follows:

1. Injection into the infra-orbital nerve through the mucobuccal fold.
2. Injection into the posterosuperior alveolar branches of the maxillary branch of the fifth nerve at the level of the second molar through the mucobuccal fold.
3. Injection into the anterior incisive canal immediately back of the space between the two central incisor teeth.
4. Injection into the greater palatine foramen, just medial to the third molar.
5. Injection into the mucous membrane of the frenum.
6. Local infiltration into the soft tissues of the lateral wall of the middle meatus.
7. Cocaine cotton pack under the inferior turbinate.

Stevenson [79] believes that intranasal surgical intervention is the operation of choice for a primary attack in all chronic infections of the maxillary sinus. Ninety-four and two-tenths per cent of 192 patients whom he operated on recovered completely following these procedures. He has found the Caldwell-Luc operation satisfactory as a radical operative measure. Transillumination and the roentgen rays are equally effective as diagnostic aids. Except in selected cases, dry treatment is effective in the after-care and is the better procedure. Contrary to popular lay opinion he considers that operative treatment for maxillary sinusitis is justified by the results. Focal disease seems to follow this type of infection. In his opinion, nasal polypi are associated with maxillary disease more often than with infection of any other sinus, and careful investigation of the maxillary sinus should be made in every case of apparent hyperplastic ethmoiditis. Conservative surgical intervention usually suffices in these cases. Sewall's method of estimating the cytologic content of the maxillary antrum seems to the author to be a distinct advance in the diagnosis of this disease. He condemns injudicious curettement of the sockets of the teeth opening into the maxillary

78. Ersner, M. S.; Pressman, J. J., and Ersner, W.: Local Anesthesia for Radical Operation on the Antrum: Improved Technic of Administration, Arch. Otolaryng. **13**:379 (March) 1931.

79. Stevenson, W.: Chronic Sinusitis: Analysis of 192 Cases That Came to Operation, Arch. Otolaryng. **13**:506 (April) 1931.

sinus. Curettement should always be followed by suitable surgical intervention to close the wound.

Shearer [80] presented a paper before a group of dental surgeons in which maxillary sinus disease with reference to dental origin was discussed. He is convinced that the number of cases of primary infections due to diseased teeth is small and that the majority of infections of the antrum of dental origin are produced by dentists who probe at the time of extraction. He considers that there is a chronic osteomyelitis of the jaw bone involving the floor of the sinus and often accompanied by pus and polyps. He advises close cooperation between dental surgeon and rhinologist and suggests a combined nasal and dental operation—a large intranasal opening made under the inferior turbinate combined with his operation of alveolectomy. This consists of the following: a reflection of the mucoperiosteum on the buccal and lingual sides of the tooth root for about one-quarter inch (0.63 cm.), the removal of all pathologic bone and smoothing of all edges of the bone, after which the flaps are returned to position and sewed securely.

Dunning [81] discussed before a group of dental men the surgical treatment of chronic maxillary sinusitis of dental origin and made a plea for close cooperation between dentist and rhinologist. He stressed particularly that no probing or curetting of the tooth socket should be employed after extraction and that no plug of any kind be placed in the opening. Instead, a "saddle plate" is advised which allows pus to flow from the sinus through the oral opening but prevents the contents of the mouth from entering the antrum. If the opening persists, the rhinologist should be called who will perform an intranasal or radical operation on the antrum at the same time that the dental surgeon performs a plastic operation on the tooth socket. The plastic operation is described in detail.

In a paper on the nasal accessory sinuses and their relation to dentistry, Bower [82] briefly describes the anatomy, signs, symptoms and treatment, especially of the antrum, and calls attention to the close relationship between this sinus and dental infection. He distinguishes between antrum disease of nasal origin and dental origin by the foul smell of those of dental origin and dental history. The author describes the probing of the antrum through dental openings.

A plea for cooperation between dentist and rhinologist is made by Spilman,[83] who describes maxillary sinusitis with reference to dental

80. Shearer, W. L.: Maxillary Sinus, J. Am. Dent. A. **18**:340, 1931.

81. Dunning, H. S.: Surgical Treatment of Chronic Sinusitis of Oral Origin, J. Am. Dent. A. **18**:1307, 1931.

82. Bower, R. L.: Diseases of the Nasal Accessory Sinuses and Their Relation to Dentistry, Internat. J. Orthodontia **17**:895, 1931.

83. Spilman, F. J.: Cooperation of Dentist and Otolaryngologist in Disease of the Antrum, J. Indiana M. A. **24**:546, 1931.

etiology. He states that often the dental origin is overlooked, and that in every case of antrum disease a thorough examination of the mouth should be made by a competent dentist.

Thies [84] discusses the subject of an antral sarcoma perforating into the eyeball in a child.

Tuberculosis of the antrum is reviewed by Fallas.[85]

Gaillard [86] discusses syphilis of the antrum.

Other authors who have contributed articles on the antrum are: Massione,[87] Cusenza,[88] Aubin and Ardoin,[89] Metzianu and Tempea,[90] Ferreri,[91] Terracol,[92] Goldmann,[93] Barajas,[94] Hauberrisser [95] and Vichot.[96]

THE SPHENOID SINUS

Pressman [97] describes a method by which shrinking solutions could be applied easily and efficiently to the spheno-ethmoidal recess when the approach to this region by way of the anterior nares was blocked by a deviated septum. He places a DeVilbiss tip bent upward slightly more than a right angle in the mouth in back of the soft palate. In this way the spray is directed upward into the proper area.

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84. Thies, O.: Antral Sarcoma Perforating into the Eyeball in a Child, Klin. Monatsbl. f. Augenh. **86**:345, 1931.

85. Fallas: Tuberculosis of the Antrum, Oto-rhino-laryng. internat. **15**:432, 1931.

86. Gaillard, R.: Syphilis of the Antrum, Oto-rhino-laryng. internat. **15**:451, 1931.

87. Massione, G.: Papilloma of the Nose and of the Maxillary Sinus, Tumori **5**:258 and 344, 1931.

88. Cusenza, G.: Direct Contact of the Infra-Orbital Nerve with the Mucous Membrane of the Antrum, Arch. ital. di otol. **42**:233, 1931.

89. Aubin, A., and Ardoin, G.: Pathologic Anatomy of Acute and Chronic Sinusitis, Oto-rhino-laryng. internat. **15**:388, 1931.

90. Metzianu, N. I., and Tempea, V.: Relation of the Antrum to Ozena, Oto-rhino-laryng. internat. **15**:448, 1931.

91. Ferreri, G.: Arterial Circulation in the Upper Jaw and Antrum in an Adult, Riv. oto-neuro-oftal. **8**:205, 1931; Oto-rhino-laryng. internat. **15**:372, 1931.

92. Terracol, J.: Anatomicosurgical Data on the Antrum, Oto-rhino-laryng. internat. **15**:337, 1931.

93. Goldmann, R.: Radical Operation on the Antrum with Drainage Through the Middle Nasal Duct, Monatschr. f. Ohrenh. **65**:28, 1931.

94. Barajas, J. M.: Occult Empyema of the Right Sinus, Siglo méd. **87**:186 (Feb. 21) 1931.

95. Hauberrisser, E.: Prolapse of the Mucosa of the Maxillary Sinus and Its Plastic Replacement, Arch. f. klin. Chir. **166**:241, 1931.

96. Vichot, J.: A Case of Plurilocular Cysts of the Antrum, Stomatol. **29**:201, 1931.

97. Pressman, J. E.: Convenient Method of Shrinking the Sphenoid Drainage Area, Laryngoscope **41**:181, 1931; discussion, ibid., p. 153.

Ciardullo [98] describes two cases in which a pistol bullet lodged in the sphenoid sinus and remained in position for three and two years, respectively. In the first case, that of a youth of 15, there was an alteration in the rate of growth of different parts of the body. The skull became extremely large in its horizontal diameter, the limbs became longer and the hands and feet larger than normal. In the second case, that of an adult, 31 years of age, spontaneous nystagmus, unsteady gait, polyuria and irritability developed. The author considers these symptoms a result of damage to the pituitary gland, first in a growing youth and secondly in an adult.

Cohen [99] devised an instrument for opening and removing part of the anterior wall of the sphenoid sinus. It consists of a small burr with a collar (to prevent too deep penetration into the cavity of the sinus) on one end of a steel probe and a rasp on the other.

PARANASAL SINUSES IN GENERAL

A study of 144 alcoholic persons was made by Dick and Colbert [100] to determine whether these patients with extensive sinusitis are more prone to hallucinations than alcoholic persons who are free from disease of the sinuses. They conclude that there did not appear to be any relation between the sinusitis and hallucinosis. They add that a primary sinusitis can no doubt cause a mental state of stupor or delirium. No greater rôle can be attributed to sinusitis in the production of hallucinosis than would be played by other acute infections or toxic agents.

Chase [101] calls attention to the failure of many rhinologists to recognize satisfactorily easily diagnosable sinus disease, and enumerated the causes therefore. He makes a plea for thoroughness, hard work and seriousness in endeavor, and states that many were not trying and were lazy. He stresses a proper source of light, antrum lavage with cytologic examination and examination with iodized poppy seed oil 40 per cent.

In a paper on the cause and relief of headache of nasal sinus origin, Arbuckle [102] divides the etiology into (1) disorders of the sinuses due to infection or allergy, (2) syphilis and (3) new growths. Impacted teeth are also included. He stresses particularly the importance of allergy in the causation of sinus disease and headache. He states that when infection is present in chronic sinus disease, it frequently is super-

98. Ciardullo, G.: Projectile Remaining in the Sphenoid Sinus for Two Years, Boll. d. mal. d. orecchio, d. gola e d. naso **49**:33, 1931.

99. Cohen, S.: Sphenoid Burr and Rasp, Arch. Otolaryng. **14**:828 (Dec.) 1931.

100. Dick, W. M., and Colbert, C. N.: Nasal Sinusitis in a Series of Alcoholic Hallucinoses, Arch. Otolaryng. **14**:327 (Sept.) 1931.

101. Chase, W. D.: Undiagnosed Nasal Sinusitis, Pennsylvania M. J. **35**:167, 1931.

102. Arbuckle, M. F.: Headache: Cause and Relief, J. Missouri M. A. **28**:579, 1931.

imposed on an allergic reaction and in the great majority of cases will clear up without any particular treatment, provided the allergic reaction is encouraged to subside by proper therapy. A number of case reports are cited.

Brown [103] makes a plea for conservatism in surgical intervention in pleural infections of the nasal accessory sinuses. He advocates careful study of each case, small repeated intranasal operations under local anesthesia and, as a last resort, the extensive "clean up" procedures.

In an article on the treatment of paranasal sinusitis, Bray [104] advocates prophylaxis such as general hygiene, out of door exercise, sunshine, diet and proper clothing. The acute sinus conditions he treats by conservative intranasal shrinking without surgical intervention. If the condition becomes subacute or chronic, it is dealt with by simple intranasal operation directed toward drainage.

In a paper on the treatment of rhinitis and sinusitis with zinc ionization, Sputh [105] attributes its unusual benefits in selected cases to its shrinking properties. He especially advocates its use following intranasal operation to restore the nasal tissue to normalcy.

Haseltine [106] points out the efficacy of nasal treatments of tampons saturated with 10 per cent colloidal metallic silver in aqueous solution applied by the method of Dowling—no shrinking spray such as cocaine or ephedrine preparatory to treatment since this is followed by a secondary swelling which defeats the purpose of the original treatment. This treatment differs from all other local applications in that it is not a surface treatment but depends for its effect on the passing of the medication through the membrane into the underlying tissue. The effect lasts twenty-four hours and produces no secondary reaction.

In an article on the relation between sinus disease and focal infection, Pond [107] concludes that sinus infection is responsible for many systemic and local diseases, especially nephritis in children and retrobulbar neuritis at any age.

Acute sinusitis is discussed briefly by Hallinger.[108] Mechanical obstructions in the nose preventing aeration and drainage from the

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103. Brown, J. E.: What Constitutes Conservatism in the Surgery of Plural Infection of the Nasal Accessory Cavities? Ohio State M. J. **27**:287, 1931.

104. Bray, E. R.: Treatment of Paranasal Sinusitis, Minnesota Med. **14**:1051, 1931.

105. Sputh, C. B.: Zinc Ionization in Nasal Sinus Disease, J. Indiana M. A. **24**:461, 1931.

106. Haseltine, B.: Colloidal Tamponing (Colloidal Silver) in Treatment of Nasal Sinus Diseases, Eye, Ear, Nose & Throat Monthly **10**:53, 1931.

107. Pond, C. W.: Infections of the Nasal Sinuses; Relation to Some Local and Systemic Diseases, Northwest Med. **30**:1, 1931.

108. Hallinger, E. S.: Nasal Sinusitis, J. M. Soc. New Jersey **28**:475, 1931.

sinuses are stressed. Infection on top of this leads to the usual turgescence and blocking of the sinus orifices with the well known signs and symptoms of acute sinusitis relieved by drops that shrink the nasal tissue and occasional puncture of the antrum.

Amano [109] treated ten persons with chronic sinusitis by local applications of autogenous vaccines and antivirus by the Proetz displacement method. Seven showed improvement with one apparent cure. The author concludes that the treatment for sinusitis and ozena by the foregoing method may constitute an improvement over present treatment.

Gill [110] discusses fractures involving the orbit and paranasal sinuses. Owing to the automobile and aeroplane, these are becoming more common. Diagnosis is made by inspection, palpation and roentgen examination. Treatment of the depressed malar and anterior face of the antrum came in for most of the discussion. Gill uses a heavy instrument resembling a towel clip, which is used to grasp the depressed malar and pull it out. If this is not strong enough, a blunt instrument in the buccal fold is used as a lever in conjunction with the clip. A curved blunt instrument in a naso-antral wall opening is used to elevate fragments of the anterior face of the antrum. Appliances are usually not necessary unless the fractures are bilateral and extensive. Early diagnosis and treatment before much swelling is apparent are strongly urged. Antitetanic serum should be administered when open wounds incurred on the highways are present.

In a report covering a period of five years of study of 386 consecutive patients with chronic arthritis, Fineman, Snyder and Traeger [111] were convinced that paranasal sinusitis played a most important part in the onset, progress and prognosis of the disease in 93 cases. One in every 4 patients of their series, therefore, had sinusitis proved by clinical and roentgen examination. They feel that the excellent clinical results obtained can be attributed in a large measure to the aid of the rhinologist. No case in their group was aggravated by operative treatment of the diseased sinuses. In most cases the sinus disease was asymptomatic or "silent."

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109. Amano, K. W.: Local Immunization Treatment of Nasal Sinus Diseases, Arch. Otolaryng. **14**:30 (July) 1931.

110. Gill, William D.: Fractures Involving the Orbit and Paranasal Sinuses, with Special Reference to Diagnosis and Treatment, Texas State J. Med. **27**:351, 1931.

111. Fineman, S.; Snyder, R. G., and Traeger, C. H.: Diagnostic and Therapeutic Measures in Chronic Arthritis: 1. Disease of the Nasal Sinus in Chronic Arthritis with Special Reference to Cases in Which It Acted as a Silent Focus of Infection, Proc. Staff Meet., Mayo Clin. **6**:695, 1931.

Hurd [112] makes the statement that avitaminosis, allergy and, probably, endocrine imbalance are the underlying causes that upset the surface tension of nasal sinus mucosa and allow the subsequent bacterial invasion of the sinuses. He believes that ethmoiditis is never present with a normal middle turbinate. Roentgenograms of the sinuses with opaque substances, in his opinion, are of no value. Many case histories of sinus disease with this treatment are reported.

Goldsmith [113] believes that persistence of discharge from any nasal sinus after operation does not necessarily demand further radical surgical measures. Too much importance has been attached to the resultant discharge and is further evidence of the extent to which the subject of foci of infection has clouded our vision.

The type of patient is of importance in determining treatment. The neurotic patient should have his fears minimized and be encouraged not to worry. Regulation of habits, diet and exercise is stressed. Many of the clinical problems of sinus disease are mentioned. He discusses the causes of failure after operations on the nasal sinuses as follows:

1. Insufficient removal of the diseased mucosa.
2. Failure to secure sufficient drainage and aeration into the nose.
3. Faults in operative technic resulting in incomplete operations.

Shea [114] feels that in the management of fractures involving the paranasal sinuses the basis of treatment is the replacement of the bony fragments and the drainage of the affected sinus. Displacement of the malar bone and depressed fracture of the frontal sinus are discussed. The treatment of the transverse fracture of the upper jaw by wiring the teeth together is given.

Schuster and Schuster [115] call attention to the great value of favorable climatic conditions on sinus disease. This they attribute to the ultraviolet rays, low humidity, absence of fog and high average temperature.

Wolf [116] believes that, though far from perfect, rhinologic surgical measures compare favorably with other operations for chronic conditions. End-results depend on the proper operator, proper judgment and proper operative procedure. He advises building up the general condition of the patient prior to operation, cooperation with internist and

112. Hurd, Lee M.: Some Aspects of Nasal Accessory Sinusitis, Pennsylvania M. J. **35**:57, 1931.

113. Goldsmith, P. G.: The Treatment of Paranasal Suppuration Persisting After Operation, Canad. M. A. J. **24**:27, 1931.

114. Shea, J. J.: Management of Fractures Involving the Sinuses, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1930, p. 21; J. A. M. A. **96**:418 (Feb. 7) 1931.

115. Schuster, Stephen A., and Schuster, Frank P.: The Climatic Factor in Paranasal Sinusitis, Texas State J. Med. **26**:657, 1931.

116. Wolf, G. D.: End Results of Rhinological Surgery, M. J. & Rec. **133**: 178 (Feb. 18) 1931.

steering clear of psychoneurotic patients. The author is convinced that certain susceptible patients with probably an inherited tendency are particularly fertile soil for disease of the upper respiratory tract and nasal sinuses.

Hill [117] stresses the importance of sinus disease to the general diagnostician because of its possible etiologic factor in focal infection, common head colds and allergic conditions. He discusses the signs, symptoms and conservative and radical treatments of acute and chronic sinusitis, and calls for common sense, surgical judgment and careful study of each case. He quotes Fenton, who considers that some of the beneficial results of radical operation may be due to the defense reaction after trauma, rather than the removal of foci alone.

The importance and prevalence of sinusitis as a complication of scarlet fever are touched on by Gowen and Gowen.[118] Early in the disease scarlet fever streptococcus toxin is administered. Shrinking nasal drops followed by antiseptic medication for promotion of drainage and aeration of the sinuses is their basis of treatment. Lavage of the antrum, removal of the anterior tip of the middle turbinate and adenoidectomy are occasionally necessary. The external operation on the frontal or ethmoid sinuses has never been found necessary in the authors' series.

Sparks [119] advocates the use of methyl piperedin (proparylbenzoate hydrochloride) instead of cocaine in nasal work and claims for it great rapidity in shrinking and no toxicity in either topical application or injection. It can be used in 10 per cent solution or in full strength with epinephrine or ephedrine. No narcotic blank is required.

Sykes and Shilvock [120] report the cases of 3 patients, inmates of a hospital for mental diseases, in whom a long-standing focus of infection, such as was found in the nasal sinuses at autopsy, probably acted as a reservoir for organisms and toxins. The elaborated toxins, while not responsible in these cases for the mental condition, acted as a focus of infection and produced an adverse influence, resulting in a case of heart failure, a kidney lesion and an alimentary condition.

In a paper on postoperative physical aids in otolaryngology, Hollander [121] states that physical methods such as infra-red radiation and diathermy have aided in minimizing postoperative discomfort, shortened the period of convalescence and elevated the standards of good end-

117. Hill, F. T.: Problem of Nasal Sinus Disease, Maine M. J. **22**:119, 1931.

118. Gowen, F. V., and Gowen, T. F.: Scarlet Fever; Nasal Sinusitis, Laryngoscope **41**:564, 1931.

119. Sparks, A. L.: Advantages of Neothesin in Radical Antral Operations, M. Arts & Indianapolis M. J. **34**:636, 1931.

120. Sykes, K. A. H., and Shilvock, W. H.: Three Cases of Nasal Sinus Disease (in Mental Patients), J. Ment. Sc. **77**:185, 1931.

121. Hollander, A. R.: Postoperative Physical Aids in Otolaryngology, Arch. Phys. Therapy **12**:19, 1931.

results. He advocates and describes in detail intranasal zinc ionization as useful in reducing mild hypertrophy of the inferior turbinate. Also, he advocates submucosal coagulation of the inferior turbinate after Beck and believes in the benefits derived from zinc ionization of antrums through an opening in the naso-antral wall.

A review of the literature on nasal sinus disease appearing between October, 1930, and March, 1931, is contributed by Joseph [122] and Vernieuwe.[123] Rebattu [124] reports a case of tuberculosis of the nasal sinuses.

Contributions to some phase of general sinus disease have been made by Palmer,[125] Jones and Kistner,[126] Vignes [127] and Klostadt.[128]

Mullin [129] states that if the time spent in instilling iodized oil were occupied in studying a well taken film with a skilled roentgenologist an equal amount of information would be obtained in the majority of cases. The author advocates conservatism in surgical treatment of the sinus and considers "complete exenteration" of the ethmoid impossible. In the surgical treatment of the frontal sinus, he prefers the external to the intranasal operation.

SINUSES IN CHILDREN

In a worthwhile abstract of recent literature on vitamins and deficiency diet in their relation to sinusitis, Harris [130] quoted Stucky, Coffin, Barlow, Shurley, and particularly Dean.

Their conclusions are:

1. A deficiency in vitamin A causes a loss of appetite, loss in body weight and infections, especially in the sinuses, ears and lungs.

2. Though vitamin A is probably responsible for damaging lymphatic changes, it is difficult to say how much is due to vitamin A deficiency and how much to secondary infection.

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122. Joseph, L.: Review of the Literature on Nasal Sinus Disease Appearing Between October, 1930, and March, 1931, München. med. Wchnschr. **78**:976, 1931.

123. Vernieuwe: Tuberculosis of the Nasal Sinuses, Rev. de laryng. **52**:159 (March 15) 1931.

124. Rebattu, J.: Tumorous Type of Tuberculosis of the Nasal Sinus: Case Report, Rev. de laryng. **52**:531 (Oct. 15) 1931.

125. Palmer, A.: Rôle of Nasal Sinus Disease in Nephritis, New York State J. Med. **31**:1069 (Sept. 1) 1931.

126. Jones, N. W., and Kistner, F. B.: Chronic Sinus Infection in Relation to Systemic Disease, Ann. Int. Med. **4**:752, 1931.

127. Vignes, H.: Sinusitis as Cause of Fever in Puerperium: Case Report, Gynécologie **30**:449, 1931.

128. Klostadt, W.: Treatment of Fresh Fractures of the Nasal Sinuses, Med. Klin. **27**:1235, 1931.

129. Mullin, W. V.: The Management of Chronic Sinus Disease, New York State J. Med. **30**:1280 (Nov. 1) 1930.

130. Harris, T. J.: Vitamins and Deficiency Diet in Their Relation to Nasal Sinusitis: Abstract of Recent Literature, Ann. Otol., Rhin. & Laryng. **40**:38, 1931.

3. Diet alone is inadequate in influencing or curing established sinus disease. Proper drainage in addition was essential for a cure.

4. Incipient nasal infections treated with a diet rich in vitamin A can be favorably impressed.

5. The value of proper diet in sinus disease, especially of children, in connection with proper local measures and hygienic conditions, is very important.

Coffin [131] agrees as to the importance of diet and vitamin content in the prevention and treatment of sinus disease and conditions of the upper respiratory tract but doubts that scientific medicine has as yet done much in the prevention of rhinologic problems in the average person.

Stein [132] reports 443 cases in children with sinus involvement in which the tonsillectomy and adenoidectomy and antrum puncture with enlargement of the opening in the naso-antral wall were done at the same sitting. The author was prompted in doing this double operation to prevent subjecting the patients to a second etherization for operative procedures on the sinus. He claims excellent results from this procedure.

DeSanctis [133] discusses diet in sinusitis in children and comes to the conclusion that the effect of diet and vitamins is indirect, probably one of lowered resistance. In his opinion a diet free from vitamin A renders the lymph glands susceptible to infection and bacterial invasion, and, therefore, he advises the inclusion of this vitamin in the treatment of conditions of the upper respiratory tract in children. However, diet cannot take the place of proper local treatment.

Four case reports of children suffering from nephrosis are given by Wimmer,[134] in which he stated that the primary etiologic factor was found in the antrums. After proper treatment for nephrosis and irrigation of the antrums, the symptoms promptly subsided. The author states that the nephrosis results from the infection in the antrums which in turn is aggravated by the nephrosis, thus constituting a vicious circle.

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131. Coffin, L. A.: Diet in Nasal Sinusitis, Ann. Otol., Rhin. & Laryng. **40**:45, 1931.

132. Stein, S.: Antrotomy in Children During Tonsillectomy, Laryngoscope **41**:97, 1931.

133. DeSanctis, A. G.: Diet in Nasal Sinusitis in Children, Ann. Otol., Rhin. & Laryng. **40**:48, 1931.

134. Wimmer, S. D.: Relation of Sinusitis to Nephrosis in Children: Study Based on Investigations at Children's Hospital, Los Angeles, 1929, Arch. Otolaryng. **13**:159 (Feb.) 1931.

Gilliland,[135] Martin and Dickinson,[136] Beatty, [137] Simmons,[138] Reisman,[139] Marantz [140] and Carmack [141] wrote on the frequent occurrence and importance of nasal sinusitis in children. All agree as to its possibility as a focus of infection and to its treatment by tonsillectomy and adenoidectomy, rest, a diet rich in vitamin A, proper hygienic treatment, exposure to an alpine lamp and intranasal drainage under the inferior turbinate.

Barnett,[142] McLaurin,[143] Russell [144] and Davis [145] have contributed articles on sinusitis in children.

OCULAR COMPLICATIONS

A study of retrobulbar optic neuritis supported by a series of 15 cases observed in private practice is contributed by H. H. Vail.[146] He favors the theory of contiguity of infection from the sphenoid sinus and cited the pathologic studies of Professor Herzog of Innsbruck as showing that the structure of the bony wall between the diseased sphenoid mucous membrane and the dural sheath is the all important determining factor. If this bone is dense, no infection in the optic nerve is apt to follow; if it is composed of cellular elements, retrobulbar neuritis is likely to follow. He makes the important observation that when the onset of blindness was acute and could be definitely stated, it was always noted after awakening from sleep, owing to the position of the head in sleep favoring gravitation of pus to the upper outer angle of the sphenoid sinus where the optic nerve is in close relation. In order to show this relationship he describes a new method of taking roentgenograms.

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135. Gilliland, O. S.: Sinusitis in Children, J. Missouri M. A. **28**:587, 1931.

136. Martin, D. D., and Dickinson, J. C.: Nasal Sinus Diseases in Children, South. M. J. **24**:659, 1931.

137. Beatty, H. G.: Intranasal and Aural Infections in Children, Ohio State M. J. **27**:31, 1931.

138. Simmons, J. D.: Sinus Disease in Infants and Children, New Orleans M. & S. J. **83**:481, 1931.

139. Reisman, H. A.: Nasal Sinusitis in Children, Arch. Pediat. **48**:283, 1931.

140. Marantz, B. C.: Importance of Diagnosing and Treating Nasal Sinusitis in Infants and Children, Arch. Pediat. **48**:365, 1931.

141. Carmack, J. W.: Nasal Sinusitis in Children, Ann. Otol., Rhin. & Laryng. **40**:515, 1931.

142. Barnett, S. E.: Sinusitis in Children, J. Michigan M. Soc. **30**:150, 1931.

143. McLaurin, J. G.: Nasal Sinus Diseases of Childhood, Dallas M. J. **17**:44, 1931.

144. Russell, D. A.: Nasal Sinusitis in Children, M. Rec. & Ann. **25**:752, 1931.

145. Davis, W. B.: Sinusitis in Children, Pennsylvania M.J. **34**:305, 1931

146. Vail, H. H.: Retrobulbar Optic Neuritis Originating in the Nasal Sinuses: New Method of Demonstrating Relation Between Sphenoid Sinus and Optic Nerve, Arch. Otolaryng. **13**:846 (June) 1931.

The sphenoid is filled with iodized oil and the patient's head held so that the upper outer angle of the sphenoid is dependent. Thus the proximity of nerve sheath and sphenoid mucous membrane is visualized.

Macpherson [147] reports many cases illustrating the status of sinusitis in ocular disorders. A different treatment was given in each case cited. However, the end-results were about equal. In some, suction was used; in some, exenteration of the ethmoid cells, antral treatment, tonsillectomy and submucous resection and in some relief was obtained without treatment—all of which throws doubt on what is the really effective treatment.

Imperatori [148] abstracted the recent literature on the present status of sinusitis in ocular disorders. He quotes Prof. R. Scheerer, who considers retrobulbar neuritis of nasal origin rare in 64,000 ocular cases examined in the past seven years, 203 of which were retrobulbar neuritis. Cushing makes a categorical denial of the possible production of choked disk by sphenoid or ethmoid lesion without intracranial pressure. McMahon states that chronic spheno-ethmoiditis is a distinct clinical entity in which the operative results are attended by a high percentage of improvement or complete recovery. This is concurred in by Fenton, who also stated that too often nasal operations are advised in which other causes for the ocular condition were responsible. Leon White is quoted as always eliminating dental and tonsillar sepsis. Beck maintains that blockage of a sinus without clinical manifestation is responsible for severe edema of the optic nerve sheath and that operations lessen this dangerous edema. Portman considers that even though the curettement of the cavities apparently shows nothing abnormal, the operation is effective through the local blood letting and aeration of cavities. Edgar S. Thomson's conclusions are summarized: Sinus diseases cause iritis, cyclitis, retinitis and choroiditis in almost as great frequency as they cause optic neuritis. The ophthalmologist should be the one to decide when the sinus operation should be done. Time is of the greatest importance, and where no other cause for the pathologic process in the eye is found, the sinus operation should not be delayed.

Crigler [149] quotes Thomson to the effect that sinusitis of a purulent nature may produce secondary foci of inflammation in any part of the eye, especially retrobulbar neuritis, and advises a prompt operation on the sinus when other etiologic factors have been ruled out. He classifies ocular conditions under two heads. First, and probably the most

147. Macpherson, D.: Present Status of Nasal Sinusitis in Eye Disorders: Rhinological Aspect, Laryngoscope **41**:451, 1931.

148. Imperatori, C. J.: Present Status of Nasal Sinusitis in Eye Disorders: Summary, Laryngoscope **41**:462, 1931.

149. Crigler, L. W.: Present Status of Nasal Sinusitis in Eye Disorders: Ophthalmological Aspect, Laryngoscope **41**:457, 1931.

frequent, is that of a plastic neuritis resulting from a purulent or low grade chronic sinusitis which extends by continuity of structures first to the covering of the nerve and thence to its substance; the second classification is that characterized by a purely functional interruption of the visual currents resulting from an obscure pathologic process. The condition is characterized by an early scotoma for colors and results in absolute blindness.

Faulkner [150] summarizes the place of the rhinologist in the treatment of ocular diseases as follows: When an orbital abscess or cellulitis complicates an acute frontal or ethmoid sinusitis, the author advocates conservative treatment by frequent shrinking of the mucous membrane of the nose supplemented by suction. If the condition does not respond, simple incision for evacuation of pus and a minor nasal operation usually suffice until a later time when more radical surgical measures are used.

On the subject of intrinsic diseases of the eye, such as retrobulbar neuritis, retinitis, choroiditis and keratitis, the author stated that there is considerable variation of opinion as to etiology. The discrepancy in opinion seems to arise from the fact that many of the sinus infections that produce ocular complications are of rather a low grade hyperplastic type without pronounced symptoms. However, most rhinologists are agreed that sinusitis is a definite etiologic factor. Teeth and tonsil infections, the author considers as the more frequent offenders. Three typical case histories are presented.

Gifford [151] reviews the relationship between sinusitis and retrobulbar neuritis and comes to the following conclusions:

1. In the causation of retrobulbar neuritis, multiple sclerosis is the most important disease, probably accounting for over 50 per cent of cases.

2. Cases due to purulent sinusitis comprise about 3.5 per cent.

3. Opinions differ as to how many cases are due to latent or hyperplastic sinusitis, probably 15 or 20 per cent of cases.

4. Where no etiologic factor can be found, exploratory operation on the sinuses is indicated.

Hyperplastic sinus disease with or without symptoms in the sinuses themselves is given by Higgins [152] as a definite etiologic factor in retrobulbar neuritis. He makes a plea for repeated mappings of the visual fields and states that without a central scotoma a diagnosis of retrobulbar neuritis cannot be made. The diagnosis once made calls for prompt operation on the paranasal sinuses.

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150. Faulkner, E. R.: Place of Otorhinolaryngologist in Treatment of Eye Diseases, New York State J. Med. **31**:1386, 1931.

151. Gifford, S. R.: Relation of Paranasal Sinuses to Ocular Disorders, Especially to Retrobulbar Neuritis, Arch. Ophth. **5**:276 (Feb.) 1931.

152. Higgins, S. H.: Hyperplastic Sinus Disease in Relation to Retrobulbar Neuritis, Illinois M. J. **59**:340, 1931.

Ford [153] reviews a case of apparent choroiditis leading to blindness which by the use of the scotometer was found really to be retrobulbar neuritis due to latent unsuspected sinusitis. No signs or symptoms pointed to the sinuses. An operation on the paranasal sinus resulted in prompt return of vision.

Smith [154] considers low grade ethmoiditis an important etiologic factor of retrobulbar neuritis and favors prompt operation on the sinuses. He feels that the toxemia or blood stream mode of transmission of the disease to the optic nerve is more probably correct than transmission by continuity.

Peters [155] discusses certain clinical types of ocular pathologic processes, such as (1) enlargement of the blind spot, (2) retrobulbar neuritis, (3) manifest optic neuritis, neuroretinitis and papillitis, (4) chiasmal and tract diseases and (5) residual optic atrophy, resulting from diseases of the paranasal sinuses, and makes a plea for prompt surgical intervention on the sinuses, even an exploratory operation when requested by a competent ophthalmologist on proper study of the case.

In a paper dealing with abducens paralysis secondary to chronic hyperplastic spheno-ethmoiditis, Horgan [156] reported a case in which all the sinuses on the contiguous side to the diseased eye were found at operation to be filled with a viscid and cloudy secretion after which the ocular condition cleared up. The case is interesting in that it lends definite support to the view that retrobulbar neuritis and oculomotor paralysis are induced by direct spread of infection from the sphenoid and posterior ethmoid cells.

Vail, Jr.,[157] reviews the orbital complications of sinus disease. He points out that two thirds of the orbital plate is of thin bone which is at the same time the partition of the nasal sinuses and which is traversed by many veins and foramina. This makes the orbital contents particularly liable to damage. The fact of prime importance is to have a rhinologic examination in all cases that do not quickly respond. The more usual orbital complications are mucocele, osteoperiostitis, diffuse orbital swelling, abscess, involvement of the optic nerve, uveitis and functional disturbances especially in the intra-ocular and extra-ocular muscles.

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153. Ford, R.: Retrobulbar Neuritis, Masked by Choroiditis and Due to Latent Nasal Sinusitis: Case Report, Proc. Roy. Soc. Med. (Sect. Ophth.) **24**:35, 1931.

154. Smith, Wallace Bruce: Low Grade Ethmoiditis as a Cause of Certain Eye Conditions, California & West. Med. **34**:88, 1931.

155. Peters, L. C.: Clinical Types of Ocular Pathology Due to Disease of the Paranasal Sinuses, New York State J. Med. **31**:267, 1931.

156. Horgan, J. B.: Abducens Paralysis Secondary to Chronic Hyperplastic Spheno-Ethmoiditis, J. Laryng. & Otol. **46**:542, 1931.

157. Vail, D. T., Jr.: Orbital Complications in Sinus Disease: Review, Am. J. Ophth. **14**:202, 1931.

In a paper on "Choked Disk," by Bulson,[158] there is quite a discussion as to whether or not a pathologic process in the sphenoid sinus could cause this condition. The author quotes many accepted authorities on both sides of the question. The controversy probably lies in the definition of the term "choked disk." Choked disk from increased intracranial pressure cannot be caused from a sphenoidal pathologic process. On the other hand, a retrobulbar neuritis, which cannot be distinguished objectively from choked disk, can definitely result from such a cause. In a questionable case the author does not delay surgical intervention on the sphenoid for too long a period.

A paper with fifty-six visual field charts, by Jones,[159] stresses the importance of routine mapping of the visual fields in cases of sudden impairment of vision.

Kramer [160] reports a case of retrobulbar neuritis caused by a removable radium emanation seed which had been allowed to remain in the nose for one and one-half years. The seed had been placed in the nose for chronic ethmoiditis but was lost and not recovered by the previous rhinologist.

A symposium on the ocular symptoms resulting from sinus disease together with its medical and surgical treatments is outlined by Buchanan,[161] Adams [162] and Champenois.[163]

Papers are also contributed by Neuschüler,[164] Behr,[165] Hughes,[166] Pérez Pórcel,[167] Russo-Frattasi [168] and Escat [169] on aspects of ocular symptomatology in nasal sinus disease.

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158. Bulson, A. E.: Choked Disk (Papilledema) Due to Diseases of the Sphenoid Sinus, J. A. M. A. **97**:926 (Sept. 26) 1931.

159. Jones, C. P.: Visual Fields in Toxic Blindness Due to Foci in the Mouth and Nose, Virginia M. Monthly **58**:316, 1931.

160. Kramer, R.: Radium Seed in the Ethmoid, Laryngoscope **41**:17, 1931.

161. Buchanan, C. C.: Eye Symptoms and Changes Resulting from Sinus Disease: Treatment, New Orleans M. & S. J. **84**:275, 1931.

162. Adams, J. C.: Medical Treatment of Nasal Sinus Diseases Complicated by Eye Disturbances, New Orleans M. &. S. J. **84**:269, 1931.

163. Champenos, F.: Surgical Treatment of Nasal Sinus Diseases Complicated by Eye Disturbances, New Orleans M. & S. J. **84**:272, 1931.

164. Neuschüler, I.: Ocular Symptomatology in Nasal Sinus Disease, Riv. oto-neuro-oftal. **8**:129, 1931.

165. Behr, C.: Relation of Isolated Retrobulbar Neuritis to Inflammatory Conditions in Accessory Nasal Sinuses, München. med. Wchnschr. **78**:1378, 1931.

166. Hughes, W. F.: Ocular Complications in Nasal Sinus Disease, Tr. Indiana Acad. Ophth. & Otolaryng. **13**:84, 1929.

167. Pérez Pórcel, E.: Rhinogenic, Retrobulbar Neuritis: Treatment; Case Report, Semana méd. **2**:1413 (Nov. 5) 1931.

168. Russo-Frattasi, G.: Retrobulbar Neuritis from Changes in the Posterior Sphenoid Sinuses, Ann. di ottal. e clin. ocul. **49**:38, 1931.

169. Escat, E.: Sphenoido-Ethmoidal Sympathoma with Origin in the Sphenopalatine Ganglion: Two Cases, Ann. d'oto-laryng., 1931, p. 828.

OSTEOMYELITIS

In a paper on sinusitis and its relation to osteomyelitis, Trible [170] briefly reviews the embryology and histology of the bony development of the sinuses and describes the pathologic process of osteomyelitis of the frontal bone. Its comparisons and differences with osteomyelitis of the long bones are noted. Why osteomyelitis of the antrum, sphenoid or temporal bone adjoining an infected mastoid should occur less often than in the frontal is asked and left unanswered, except that possibly the stripping or elevation of the periosteum over the frontal sinus might be a factor. Typical case reports of osteomyelitis complicating frontal sinusitis are cited by the author.

Hastings [171] reports two cases of acute frontal sinusitis complicated by osteomyelitis of the anterior wall of the sinus in which the conservative treatment of making an opening in the floor for drainage was done at first operation. Both patients recovered. He firmly believes in the great value of leaving unmolested the periosteum over the anterior face of the frontal sinus. He makes a plea for a two-stage operation, the second to be as radical as need be.

Houser [172] presents a case report of an acute frontal sinusitis complicated by osteomyelitis of the anterior and posterior wall of the sinus with extradural abscess. Operation with removal of the anterior wall of the sinus and most of the posterior wall evacuated a large extradural abscess. The wound was packed wide open. The patient recovered.

Friesen [173] reports an acute osteomyelitis of the skull following an intranasal operation on the ethmoid sinus. The operation was directed toward keeping within the infected osteomyelitic area. The patient recovered.

Bareichnikoff [174] presents two cases of frontal sinusitis followed by osteomyelitis of the frontal bone. Emiliani [175] presents a similar case.

Furstenberg [176] has written a thorough and instructive paper on osteomyelitis of the skull based on material from fourteen cases that came

170. Trible, G. B.: Nasal Sinusitis from the Viewpoint of Osteomyelitis, Laryngoscope **41**:332, 1931.

171. Hastings, H.: Osteomyelitis Associated with Frontal Sinusitis: Value of Preserving the Anterior Wall with Attached Periosteum, Arch. Otolaryng. **13**:181 (Feb.) 1931.

172. Houser, K. M.: Acute Sinusitis Complicated by Osteomyelitis of the Frontal Bone and Extradural Abscess: Recovery, Laryngoscope **41**:504, 1931.

173. Friesen, J. F.: Recovery of Acute Osteomyelitis of the Skull Following Operation of the Frontal Sinus, Laryngoscope **41**:19, 1931.

174. Bareichnikoff, J.: Diseases of the Frontal Sinus Followed by Osteomyelitis of the Frontal Bone: Two Cases, Ann. d'oto-laryng., 1931, p. 642.

175. Emiliani, C. M.: Diseases of the Nasal Sinus: Case with Frontal Osteomyelitis, Rev. de especialid. **6**:830, 1931.

176. Furstenberg, A. C.: Osteomyelitis of the Skull, Tr. Am. Laryng., Rhin. & Otol. Soc., 1931, p. 1.

under his personal supervision. The etiology of the condition he considers still unsettled. The frequent occurrence after an acute frontal operation he feels is due to surgical trauma plus lowered resistance. The disease spreads by two different pathologic pathways: continuity of tissues, and hematogenous metastases by way of thrombophlebitis of the intracranial diploic veins.

The author states that the external periosteum has little to do with the life of the cranial bones, that the dura furnishes most of the nourishment to the skull cap and assumes an interesting rôle in osteogenic repair. A detailed description of the pathologic process in the bone and the mode of spread follows. The only hope of success in treatment is radical surgical removal of the diseased bone. The author considers that the suture lines form a barrier to the spread of infection and tries to limit the bone removal to this point. In the series of fourteen patients there are eight living and well with complete bony regeneration of the skull. One of these patients had previously had almost all of the calvarium removed.

Wolcott [177] reports a case of acute osteomyelitis and frontal lobe abscess complicating an acute frontal sinusitis. The author does not give any details regarding his technic in removing the osteomyelitic bone but merely states that after removal of the diseased bone and repeated aspiration of the abscess the patient made a complete recovery.

ROENTGENOLOGY

A history of the development of transillumination and roentgenography of the maxillary sinus together with their relative merits is reviewed by Gafafer.[178] Many divergent opinions are held by prominent rhinologists in comparing these two methods. Some greatly favor transillumination, stating they find roentgenograms unnecessary as they usually give no additional information. On the whole, many more are enthusiastic for the final details of roentgenography and consider it indispensable for proper diagnosis. The real test in transillumination is to get the light reflected through the pupil. The failure in roentgenography is often due to poor x-ray plates, poor technic and, what is more important, poor interpretation.

Graham Hodgson [179] describes a new standardized technic for roentgenography of the sinuses. The author considers that the chief cause

177. Wolcott, C. C.: Acute Frontal Sinusitis, Orbital Cellulitis, Osteomyelitis and Abscess of the Brain with Recovery, Arch. Otolaryng. **14**:476 (Oct.) 1931.

178. Gafafer, W. M.: Transillumination and Roentgenography of the Antrum: Review, Arch. Otolaryng. **14**:737 (Dec.) 1931.

179. Graham Hodgson, H. K.: Radiography of the Accessory Nasal Sinuses: New Standardized Technique for Exact Projection of All Sinuses, Together with Demonstration of Fluid Levels, Brit. J. Radiol. **4**:421, 1931; Nasal Sinusitis and Its Radiological Demonstration, Proc. Roy. Soc. Med. (Sect. Laryng.) **24**:103, 1931; J. Laryng. & Otol. **46**:729, 1931.

for poor interpretation of sinus plates is faulty technic. He uses a technic, employing an upright position to show fluid levels in the antrum and maintains that the positions used must be absolutely standard for all patients; that is, each position in every case must be exactly the same. To accomplish this he uses an imaginary line connecting the external canthus of the eye and the auditory canal.

Brown [180] discusses the surgical and pathologic significance of roentgenograms in diseases of the maxillary antrum. He quotes Hajek in basing his interpretation of roentgenograms on the variation in the "air shadow." The small sinus has thick walls and a small volume of air, whereas the large cavity has thin walls and a large volume of air. The larger the cavity the more intense will be the air shadow.

A thorough discussion of each of the following subdivisions, amply illustrated with roentgenograms, follows:

1. Conditions in which there were alterations in the bony wall—sarcoma, carcinoma, penetrating foreign bodies, dental cysts, dentigerous cysts, acute and chronic osteitis with their subdivisions, osteoma and conditions following operations on the maxillary sinus.

2. Conditions in which there are alterations in the contents of the bony cavities —acute and chronic inflammatory changes, cysts, hydrops, calculus, cholesteatoma and new growths.

Allen's [181] paper on ordinary pathologic conditions of the paranasal sinuses is amply illustrated with x-ray films. He states that for the roentgenologist to become proficient in interpreting pathologic processes in the sinus he must be present at the operation and there correlate his roentgen shadows with the pathologic process actually found.

Law,[182] in a paper on proper interpretation of roentgenograms of the sinus, makes a plea for standardized technic. He insists on stereoscopic lateral views in order to show changes in the walls of the ethmoid septums. He prefers a film of less density and contrast to show these minute bony changes. The use of nonopaque fluid he advises in selected suitable cases but not as a routine procedure. He asks that the three capital Cs be heeded to get the best result from the roentgenologist: cooperation, consultation and coordination.

Womack and Hume [183] advise the use of 40 per cent neosilvol in a lubricating jelly base (surgical lubricant) instead of iodized oil in sinus-

180. Brown, H. G.: Skiagrams in Diseases of the Maxillary Sinus: Surgical and Pathological Significance, J. Laryng. & Otol. **46**:670, 1931.

181. Allen, B.: Roentgenographic and Clinical Observations of Nasal Sinuses, Am. J. Roentgenol. **26**:214, 1931.

182. Law, F. M.: Interpreting Nasal Sinus Roentgenograms, Ann. Otol., Rhin. & Laryng. **40**:82, 1931.

183. Womack, D. R., and Hume, J. R.: Neosilvol (Silver Iodide Preparation) in Sinusography, Laryngoscope **41**:203, 1931.

ography, and claim for it the following advantages: (1) It is a perfect medium for x-ray work. (2) The roentgenogram need not be taken immediately since the material remains in the sinuses without spilling. (3) The neosilvol is more pleasant to taste than iodized oil, (4) does not stain the clothing, (5) costs less and (6) is easily obtainable. (7) It has a decided therapeutic effect that iodized oil does not possess. (8) There is no danger of argyrosis.

Beeler, Smith and Collins [184] prepared a paper amply illustrated with x-ray films wherein they strongly advised the use of opaque oil (iodized oil) in visualizing actual pathologic processes in the maxillary antrum. They call attention, however, to a swelling reaction of the mucous membrane which lasts until the oil has been extruded, and which may on a subsequent roentgen examination reveal a picture not consistent with actual pathologic process.

Schneck,[185] Dean and Morsman,[186] Richards [187] and Hasty [188] wrote on the value of good roentgenograms properly interpreted, and cited the advantages of the use of iodized oil.

Potter [189] describes in detail the grid diaphragm principle applied to roentgenography of the paranasal sinuses. In taking roentgenograms with this method the patient is in a sitting position with head bowed slightly forward, the apparatus is arranged for accurate centering and immobilization of the head and the exposure is made on the smallest film area within reason to keep down the size and weight of the grid apparatus.

Mittermaier [190] describes a method of roentgenography of the sinuses for which he claims simplicity, ease of operation and clearness in outline. The patient is comfortably seated in an upright position before a tube, which can be inclined in any position, which allows the clear visualization of all parts of the sinuses. The author does not think iodized oil is necessary for roentgenography of the sinus.

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184. Beeler, R. C.; Smith, L. A., and Collins, J. N.: Use of Opaque Oils in the Diagnosis of Maxillary Sinus Disease, Am. J. Roentgenol. **26**:202, 1931.

185. Schneck, N.: Lipiodol (Iodized Oil) as an Aid in Diagnosis and a Guide in Treatment of Antrum Disease, Wisconsin M. J. **30**:914, 1931.

186. Dean, A. C., and Morsman, C. F.: Lipiodol (Iodized Oil) as a Diagnostic Aid in Roentgenography of Chronic Nasal Sinus Disease, Laryngoscope **41**:196, 1931.

187. Richards, G. E.: Chronic Infections of the Maxillary Antra in Systemic Disease: A Radiological Study, Brit. J. Radiol. **4**:120, 1931.

188. Hasty, F. E.: Roentgenography of Nasal Sinuses, Laryngoscope **41**:747, 1931.

189. Potter, H. E.: Grid Diaphragm Principle Applied to Roentgenography of the Paranasal Sinuses, Am. J. Roentgenol. **25**:814, 1931.

190. Mittermaier, R.: Roentgenographic Methods in Nasal Sinus Affections, J. Laryng. & Otol. **46**:661, 1931.

Articles relative to roentgenology in nasal sinus disease have been contributed by Traina,[191] Champeil and Tillier,[192] Chaumet,[193] Broste,[194] Bouchet, Laquerriere and Deliencourt,[195] Nicotra, [196] Blassingame,[197] Whitney and Doub,[198] Robb,[199] Blocher [200] and Tyler.[201]

PULMONARY COMPLICATIONS

Hall and Thomas,[202] in a preliminary report, call attention to the symptom complex of bronchosinusitis characterized clinically by intermittent nasal blocking with or without nasal discharge, mild cervical adenopathy and an associated cough with a slightly raised inconstant afternoon fever. The tracheobronchial tree becomes involved through aspiration of infected material from the nose, by the lymphatic system or through the blood stream. Bronchiectasis may result.

Deweese [203] also contributes a paper on bronchosinusitis. Bronchosinusitis is described as a simultaneous infection of the sinuses and bronchial tree, the sinus infection causing the pulmonary pathologic process by bronchogenic or circulatory routes. The persistence of paranasal infections produces characteristic changes manifested by bronchiectasis and changes in the pulmonary field. The necessity of attention to

191. Traina, S.: Roentgen Examination in the Diagnosis of Sinusitis, Boll. d. mal. d. orecchio, d. gola e d. naso **49**:105, 1931.

192. Champeil and Tillier, R.: Roentgenotherapy in Frontomaxillary Sinusitis, Bull. et mém. Soc. de radiol. méd. de France **19**:420, 1931.

193. Chaumet: Images radiologiques d'ethmoidite, Arch. d'electric. méd. **39**:45, 1931; Radiographic Examination of the Ethmoid Sinus for Disease, J. de radiol. et d'électrol. **15**:337, 1931.

194. Broste, K.: Modification of Roentgen Diagnosis of Nasal Sinus Disease, Ugesk. f. læger **93**:589, 1931.

195. Bouchet; Laquerriere, and Deliencourt: Radiographie de l'ethmoide, Arch. d'électric. méd. **39**:39, 1931.

196. Nicotra, A.: Reactions and Radiologic Indications of Nasal Sinus Disease, Riv. di radiol. e fis. med. **3**:1, 1931.

197. Blassingame, C. D.: Standardization with X-Ray Versus Dogmatism in Diagnosis of Nasal Sinus Disease, J. Tennessee M. A. **24**:352, 1931.

198. Whitney, E. L., and Doub, H. P.: Further Studies in Use of Iodized Oil (Lipiodol) in the Diagnosis of Maxillary Sinus Disease, with a Report of 225 Cases, J. Michigan M. Soc. **30**:72, 1931.

199. Robb, J. M.: Chairman's Address (Sect. Otolaryng.), J. Michigan M. Soc. **20**:1, 1931.

200. Blocher, J. D.: Head Clamps for Fenestrated Shield in Roentgenography of the Nasal Sinus, Radiology **16**:554, 1931.

201. Tyler, A. F.: The Interpretation of Roentgenograms of the Mastoids and Nasal Accessory Sinuses, Arch. Phys. Therapy **12**:237, 1931.

202. Hall, S. S., and Thomas, H. V.: Broncho-Sinusitis: A Preliminary Report, West Virginia M. J. **27**:162, 1931.

203. Deweese, E. R.: Bronchosinusitis: Roentgen Ray Examination; Correlation with Clinical Symptoms, J. Missouri M. A. **28**:108, 1931.

sinus infection in order to relieve chronic pneumonitis and prevent incurable bronchiectasis is stressed by the author.

Osmond [204] contributes a paper on the interrelation between chronic sinusitis and pulmonary changes such as chronic bronchitis, bronchial asthma, bronchiectasis and tuberculosis. He maintains that sinus infection may be an etiologic factor in these conditions and cites case histories before and after operation with cures to substantiate his contention.

Dr. G. B. Fred of Boston assisted in abstracting this review.

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204. Osmond, J. D.: Nasal Sinus Infection in Suspected Pulmonary Tuberculosis, J. A. M. A. **97**:1778 (Dec. 12) 1931.

Abstracts from Current Literature

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LABYRINTHIC LESION WITH OTITIS MEDIA ACUTA AND MASTOIDITIS IN A PATIENT WITH CHRONIC LYMPHATIC LEUKEMIA: CASE. A. PAGANO, Arch. ital. di otol. **43**:284 (May) 1932.

In Pagano's case, a woman, aged 48, of Portici, had an attack of influenza, complicated with acute otitis and mastoiditis, and probably by a rather grave general septicemic state. To these conditions the postoperative behavior of the patient was doubtless due. Patients with leukemia in general, and particularly with lymphatic leukemia, are less resistant than usual to acute infections. When admitted to the clinic, the patient had for twenty years had chronic obstruction of the right nostril. She had a cardiopathy, and complained of excessive fatigue, edema of the lower limbs and frequent dyspnea on effort. The previous month, she had had an attack of influenza with fever, which confined her to bed for several days. During that period, she had an acute localization in the buccal cavity accompanied by marked involvement of the submaxillary glands and the pharynx. A few days after getting up, she noted suddenly a feeling of becoming deaf and of loud sounds in the right ear. She had dizziness and vomiting, accompanied by notable diminution of hearing in both ears, although more marked in the right. Because of the dizziness she went back to bed, where, since the slightest movement brought on a dizzy attack, she was obliged to remain quiet in a dark room. After about a week, during which the dizziness and the noises in the ear decreased slightly, violent pains developed in the right ear; also a feeling of tension, with pulsation but without emission of pus. Five days later, a spontaneous pain and swelling became evident in the corresponding mastoid region, while the dizziness and vomiting became worse. Other methods failing to bring relief, operation was resorted to. Under local anesthesia, owing to the cardiac condition of the patient, operation on the right mastoid region was carried out. The antral region presented a more necrotic aspect as the operator pushed farther and the tissues were easily attackable with a gage. During removal of the bone, the lateral sinus was injured and blackish blood exuded, which was readily checked by a tampon. A small portion of the dura of the median cranial fossa was laid bare, and it also was covered with blackish necrotic bone. The postoperative course was fairly good. The nystagmus and vomiting improved, but the general condition remained unchanged, and there was no improvement in audition. One had to raise the voice considerably to make the patient understand. As the septic condition persisted, a second operation was performed without anesthesia. The lips of the wound were freshened by removal of about 0.5 cm. of necrotic tissue before healthy tissue was reached, without any signs of pain on the part of the patient. The removed tissue was preserved for histologic examination. The surrounding healthy tissue presented a whitish, fatty, anemic aspect, and gave rise to considerable bleeding. The blood was very fluid, had few corpuscles and was watery and clear. The wound was enlarged, a larger portion of the sinus being laid bare. A few hours after the operation, the patient ate with considerable appetite; the same general conditions continued. At the request of the relatives, the patient was dismissed from the clinic, and it has not been possible to follow her history.

EDITOR'S ABSTRACT.

OLD PERFORATIONS OF THE TYMPANIC MEMBRANE AND THEIR TREATMENT. O. BÉNESI, Monatschr. f. Ohrenh. **66**:27 (Jan.) 1932.

Bénesi describes a new method of inducing regeneration of the tympanic membrane in old perforations of the membrane. A small plug of cotton soaked with borated alcohol is introduced into the perforation so that it lies against the

edges but does not touch the medial wall. The alcohol destroys the epithelial border of the perforation, and the foreign body irritation and hyperemia produced by the cotton after evaporation of the alcohol stimulate the tissue to proliferation. To avoid irritation of the middle ear, the cotton should not be allowed to touch the mucous membrane of the middle ear, but when this is impossible, the alcohol may be considered an adequate safeguard against infection. The cotton is renewed daily until the perforation is closed. When the margin of the perforation is much thickened, or when the destruction is extensive, a careful initial cautery with trichloracetic acid is advised, to be followed by the treatment described. The author states that this method is not advisable in chronic suppuration with osteitis, tuberculosis and syphilis, or in cases with symptoms of irritation of the labyrinth, but it is sometimes suitable in tube infections, if it is possible to stop the suppuration for a long enough period. The importance of bringing about an early regeneration of the tympanic membrane in children with dry perforations is pointed out. The author has usually had good, and never unfavorable, results with this method.

EDITOR'S ABSTRACT.

LITTLE HEEDED COMPONENT OF DEFICIENT SPEECH-HEARING IN BRADYCARDIA: SIGNIFICANCE IN AUDITORY EXERCISES. E. FRÖSCHELS, Monatschr. f. Ohrenh. **66**:454 (April) 1932.

Fröschels says that the understanding of speech depends on the hearing of speech sounds, and this function depends on two components, the perception by the peripheral sensory organ of all partial tones which constitute a speech sound and the unification or coordination of these partial tones into a complex, that is, a speech sound, by the brain centers. The former component is not given much consideration in auditory exercises for persons with bradycardia. If the peripheral sensory organ due to malformation or injury is incapable of perceiving all the partial tones of a speech sound to the same degree, or if certain tone pitches are missed entirely, the individual hears speech in a qualitatively modified form. If this qualitative change exceeds certain limits, he can no longer identify the sound with memory of words, and systematic auditory exercises must be instituted to correlate the speech in its new acoustic form with the previously learned speech. The individual must learn to identify the modified speech sounds with the old acoustic impressions. Alphabet pictures are used with adults, and with analphabetic children well known objects are employed. The increased understanding of speech manifests itself like increased hearing of speech; the patient repeats text-words correctly at greater distance than previously. The author says that partial tones or combinations of tones other than those normally used can be centrally coordinated into a meaningful complex. To make this complex linguistically utilizable is the purpose of the auditory therapy; as it concerns acquisition and exercise of a central capability, this therapy is successful in the presence of partial hearing capacity, without regard to existing organic defects.

EDITOR'S ABSTRACT.

OTOGENOUS GENERALIZED INFECTION IN NURSLINGS. I. HOFER, Monatschr. f. Ohrenh. **66**:462 (April) 1932.

Following an introductory discussion of the differentiation of various types of generalized infection, Hofer reports a case of otogenous sinus thrombosis with toxemia, in a nursling of 10½ months, which he was able to cure by surgical exclusion of the infectious focus. The infant was hospitalized because of eczema, influenza, rhinitis, bronchitis and bilateral otitis media. About three weeks later, after a slight amelioration of some of the disturbances, the otitis on the right side became acute, giving rise to mastoiditis. Neither antrotomy nor exposure and resection of the sigmoid sinus performed two weeks later were able to end the high intermittent fever, and it was only after ligation of the right jugular vein and exclusion of the septic focus, constituted by the thrombus in the sigmoid sinus, from the circulation that the temperature dropped and a marked improvement

in the general condition took place. The author designates this condition of septic generalized infection as toxemia because no bacteria could be demonstrated in several cultural examinations of the blood, and therefore it could not properly be called a bacteremia. He does not call it a pyemia, although the intermittent fever curve was characteristic of pyemia, because no metastases occurred. The author says that otologists must keep in mind that the occurrence of otogenous generalized infection, whether pyemic or of some other form, is not as rare in nurslings as many pediatricians believe. Usually these cases have a rapid fatal termination because the nurslings cannot resist a septic process of long duration, especially when the location of the septic focus, as in otogenous cases, is not diagnosed and not surgically excluded.

EDITOR'S ABSTRACT.

HEARING CAPACITY IN MAN 116 YEARS OF AGE. S. KOMPANEJETZ, Monatschr. f. Ohrenh. **66**:470 (April) 1932.

Examination of the hearing capacity of a man, aged 116 years, leads Kompanejetz to the statement that the clinical picture of presbycusis is not uniform. This case was characterized by: (1) a shortening of bone conduction, especially for higher tones, (2) a restriction of both limits of hearing and (3) a uniform reduction of auditory acuity for all tones of the tone scale. The author thinks Lederer's statement that the characteristic signs of presbycusis are the lowering of the upper limit of hearing, and decrease in perception of high tones should be revised to include also a uniform decrease in acuity for all tones of the tone scale and a restriction of both the upper and lower limits of hearing.

EDITOR'S ABSTRACT.

CLINICAL DIAGNOSIS OF INTRANASAL MENINGOCELE. O. HALLERMANN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:413 (May 13) 1932.

In connection with his personal experience with a case of intranasal meningocele in which exploratory puncture (just as in seven of the eight cases reported in the literature) produced fatal meningitis, Hallermann presents some diagnostic symptoms which he hopes will help clinicians to diagnose these cases without the dangerous intervention of puncture or biopsy. In cases of intranasal meningocele the hernia opening is in the region of the lamina cribrosa. The hernia protrudes through the opening in the ethmoid into the nose, pushing the mucous membrane of the roof of the olfactory fissure and the nasal septum before it, forcing the septum to the opposite side and pushing the ethmoid and median turbinate of the same side in a lateral direction. Rhinoscopy reveals a tumor-like downward protrusion covered with mucous membrane which more or less fills the interior of the nose; it is not pedunculate but rather continuous with the mucous membrane of the septum, at most permitting introduction of a probe between it and the lateral nasal wall. Another diagnostic characteristic of the tumor is its elasticity, tested by light pressure with a probe, and possibly pulsation. Increase of the brain pressure by compression of both jugular veins results in a loss of elasticity and increased hardness of the tumor. One or all of these characteristics should permit a meningocele to be differentiated from: polyps, which are usually movable and can be felt on all sides by a probe; fibromas, which are of tougher consistency, bleed easily at the touch of a probe and do not change consistency at increase of brain pressure, and finally malignant tumors, which usually have an uneven surface, are ulcerated and appear in persons of advanced age. Certain aspects of the facial portion of the skull may also give support to the diagnosis of meningocele: (1) widening of the root of the nose, (2) eyes unusually far apart and (3) other congenital disturbances, such as facial clefts or epicanthus. If the hernia is small and pedunculate it can be surgically exposed from the exterior and ligated, but in the majority of cases there is no pedicle and intervention is out of the question.

EDITOR'S ABSTRACT.

DEGENERATIVE ATROPHY OF THE LABYRINTH IN PATIENTS WITH TUMORS DISTANT FROM THE EAR. H. BRUNNER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**: 443 (May 13) 1932.

Brunner's interpretation of the results of anatomic and clinical examination of the ear in a woman with adenocarcinoma of the pylorus differs from Démétriades' interpretation of similar findings in cases of tumors distant from the ear. Clinically the latter found a functional decrease consisting in progressive bradycardia and labyrinthine hypo-excitability, sometimes amounting to caloric nonexcitability and anatomically a degenerative atrophy of the peripheral sensory organs, all of which he attributes to the action of toxins emanating from the tumor. In the case reported by the author cochlear examinations could not be made, but caloric reflexes could not be elicited by irrigation with small or large quantities. The extensive degenerative changes in the neuroganglion revealed at necropsy the author attributes primarily to arteriosclerotic changes, although he admits the possibility of toxic influences from the cancer. Démétriades listed the following pathologic changes in the labyrinth in his cases: (1) distention and absence of sensory organs of the maculae and christae, (2) atrophy of nerves in the maculae and christae, (3) accumulation of lipoid pigments in the cells of the vestibular ganglion and (4) slight atrophy of the main nerve trunk of the labyrinth. The first and third of these the author thinks can scarcely be attributed to a pathologic condition, and the fourth could not be demonstrated in his own case. So far as the second is concerned the author's case exhibited, in addition to probable absence of some nerve fibers, slight degenerative changes in the nerve fibers of the christae and maculae which were still preserved. The author admits that the degenerative atrophy of the labyrinthine sensory organs could result from the toxic influences of the cancer, but he does not think that the slight degeneration of some of the nerve fibers in the christae and maculae is sufficient anatomic basis for the caloric nonexcitability in these cases. The individual sensory cells of the maculae and christae are supplied by several nerve fibers so that in case some of them were destroyed the sensory cells could still react. The author considers the loss of reflex in response to caloric stimulation in his case the result of the long, severe cachexia. He cites the loss of reflexes without demonstrable disease of the brain in severe hemorrhages, poisoning and other conditions.

EDITOR'S ABSTRACT.

CALORIC VESTIBULAR REFLEXES AND NERVOUS CENTERS. M. H. FISCHER and E. OLDBERG, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:499 (May 13) 1932.

Fischer and Oldberg's investigations of vestibular reflexes show that these are dependent on the condition of the nerve centers. Through simultaneous aqueous irrigation of both ears, under certain conditions, pulsion reflexes are evoked, the duration of which can be exactly determined and proves to be constant under parallel test conditions. When such double irrigations are repeated in a continuous series with ten minute intervals the pulsion reflexes undergo peculiar changes in the course of the test series. They become shortened and weakened or lengthened and strengthened; sometimes no reflex occurs. The kind of modification depends on the irrigation constants and the irrigation intervals; often in a long test series lengthening and shortening alternate rhythmically. To explain these changing effects trial tests were undertaken by the authors. A so-called disturbance irrigation was employed to produce pulsion reflexes; then a certain test irrigation (the exact response to which has been determined in previous experiments) was given ten minutes following the disturbance irrigation, and the pulsion reflexes were observed. This combination of disturbance irrigation and test irrigation was tried at other sittings with intervals of twenty and thirty minutes and longer. In these experiments a modification of the test reflexes was seen depending on the time relation of the test irrigation to the disturbance irrigation. This can be represented graphically in a curve and exhibits a rhythmic pendulating decrease. Experiments were performed to prove that these peculiarities were not dependent on changes in the condition of the receptive organ or on vascular reactions. The

changes in the test reflexes are provoked solely by disturbance reflexes. They are based on changes of long duration in the excitability of the nervous reflex centers which are caused by the disturbance reflexes. This shows that vestibular reflexes have long-lasting after-effects on the nervous system. Clinically this means that in functional examinations vestibular stimuli may be employed only with large time intervals if one wishes to avoid gross deceptions.

EDITOR'S ABSTRACT.

STUDY OF DECREASE IN VESTIBULAR EXCITABILITY BY CALORIC NYSTAGMUS. F. WOLETZ, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:524 (May 13) 1932.

By means of evaluation of nystagmus Woletz has made a quantitative study of possible after-effects of vestibular stimuli on the central nervous system. With the help of a series of physically identical irrigations of small volume, a standard value for the rapidity and duration of the nystagmus for each subject was determined which represented the reaction of that subject to a stimulus of known quantity and quality. The effects of a test irrigation at varying time intervals after a disturbance irrigation were noted. At first the disturbance and the test stimulus were tried on the same ear, and in a later series each was tried on a different ear; in all tests a deviation from the predetermined normal reaction was revealed. As a peripheral after-effect of long duration is impossible, the only explanation for these results is a change in the condition of the nerve centers. Clinically this shows the importance of longer intervals between vestibular examinations to avoid reciprocal influences.

EDITOR'S ABSTRACT.

PATHOGENESIS OF TUBERCULOSIS OF THE MIDDLE EAR. G. THEISSING, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **22**:328 (April) 1932.

After a general discussion of the question of the pathogenesis of tuberculosis of the middle ear, including a review of the literature, Theissing reports his own investigations on this subject. Examinations of twenty-nine temporal bones of nineteen children and young adults who died of generalized tuberculosis gave evidence of the hematogenous origin of the tuberculosis of the middle ear which was found to exist in all but one of the cases. Tuberculous foci in the bone marrow occurred almost regularly, especially in children. In the majority of cases the bone marrow was the only seat of tuberculosis of the middle ear. One of the chief arguments against a hematogenous development of this disease, that an isolated tuberculosis of the bone of the mastoid process had never been observed, was thereby nullified. Four cases in which tuberculous changes of the bone marrow existed without the slightest manifestation of any disease of the mucous membrane of the middle ear are especially significant. Brieger's argument against the hematogenous route of infection, that the middle ear is not often affected in general miliary tuberculosis, is refuted by the finding in these cases, most of which showed a more or less extensive miliary dissemination. The assumption of the hematogenous origin of tuberculosis of the middle ear based on these observations was given further support by animal experiments. The author produced tuberculous infection of the middle ear and bone marrow of the temporal bone in rabbits and guinea-pigs by injection of virulent tubercle bacilli into the common carotid artery. The use of coarse emulsions of cultures of tubercle bacilli is essential for producing infection by this method. Hitherto the only experimental method of producing tuberculous infection of the middle ear was by direct infection of the tympanic membrane which is rare in the spontaneous development of the disease; the author's method uses the route that is of most importance from the modern point of view. The tuberculous changes in the ears of the animals so infected were similar to those in the human cases reported. The tuberculous manifestations usually appeared in the mucous membrane and the bone marrow at the same time and equally, or sometimes in the latter alone, rather than starting in the upper layers of the mucous membrane and gradually extending

downward to the bone as in previous experiments. The experiments, besides supporting the assumption of the otogenous origin of tuberculosis of the middle ear, prove that the notion that the primary localization of tuberculosis of the ear is exclusively in the mucous membrane is no longer tenable.

EDITOR'S ABSTRACT.

Pharynx

THE TONSILLECTOMY PROBLEM. J. COLEMAN SCAL, M. J. & Rec. **135**:123 (Feb. 3) 1932.

According to Scal, it is the duty of the laryngologist to operate on any patient who, he conscientiously believes, would be benefited by the removal of the tonsils. The dangers of surgical tonsillectomy at present are negligible, except those which exist from any operative procedure. Careful anesthesia should be the rule, and postoperative hemorrhage should be avoided by not permitting the patient to leave the operating room until the fossae are entirely clean and dry. Surgery should be relied on unless tonsils and other lymphoid tissue in the nose and pharynx can be eliminated by a less unpleasant process. Electrocoagulation is useful when tonsillar tabs and lingual tonsils are left *in situ*. Radium in the form of radon seeds is useful in inoperable cases. Desiccation, fulguration and roentgen treatments for the elimination of the tonsils are not recommended and will soon be relegated to the past. WILLIAMSON, New Orleans. [AM. J. DIS. CHILD.]

EPIDEMICS OF SEPTIC SORE THROAT OF MILK ORIGIN; PRESENT STATUS OF BACTERIOLOGY AND EPIDEMIOLOGY. D. J. DAVIS, J. Bact. **23**:87 (Jan.) 1932.

From the author's studies of septic sore throat, the following points, which are quoted in full, seem to be established: "1. The epidemics are sudden, sharp and severe, indicating a massive dosage of the infecting agent.

"2. The sick are largely limited to users of one milk supply. There are relatively few contact infections.

"3. Epidemics rapidly recede when the infected milk supply is cut off or the milk pasteurized.

"4. Usually one cow is found with udder infected with an encapsulated hemolytic streptococcus. This organism produces large mucoid, watery, spreading colonies growing especially well on ascites blood agar. These organisms were observed in the Chicago epidemic of 1910-1911 and have become known since then as the Str. epidemicus.

"5. Experiments indicate that when these streptococci are implanted in the cow's teat, they will rapidly ascend the duct and localize in the udder, causing mastitis. They may continue there for long periods thus giving rise to the carrier state. The udder may or may not reveal gross physical changes.

"6. Similar encapsulated streptococci are uncommon in the throats of normal persons (less than 1 per cent) but more common in infected throats (12 per cent of tonsils). Sporadic cases of septic sore throat indistinguishable from epidemic cases are occasionally seen. From such throats encapsulated streptococci of the epidemicus type may be found.

"7. These encapsulated streptococci produce toxins which give rise in animals to specific neutralizing antiserum. The toxins cause specific skin reactions in susceptible persons (differing from scarlet fever toxin). A study of several strains indicates that they are heterogeneous by the agglutination test. Agglutination tests however are often unreliable for encapsulated organisms. We have not as yet compared the toxin antitoxin properties of a number of different strains.

"8. From a study of many epidemics of septic sore throat, it appears that not infrequently cases of scarlet fever and erysipelas occur as complications. There is marked variation in this respect. At times scarlet fever and skin rashes are

relatively common. Again they may be rare or absent and erysipelas relatively common. On the other hand it is well known that sore throat without rash is common during epidemics of scarlet fever.

"9. Strains of streptococci classed as scarlet fever or erysipelas organisms on the basis of skin and other tests at times develop capsules and grow quite like strains of Str. epidemicus. Such strains may be the cause of milk-borne scarlet fever. The variability in these clinical manifestations together with the variability in properties of streptococci from epidemics of septic sore throat, scarlet fever and erysipelas suggest the possibility of a variable toxigenic and serologic property.

"10. A study of the world distribution of epidemics of septic sore throat of milk origin reveals three general localities; namely, (*a*) Great Britain, (*b*) Scandinavia and Denmark and (*c*) United States and Canada. In other parts of the world the methods of treating milk supplies may explain their freedom from infections."

STOESSER, Minneapolis, Minn. [AM. J. DIS. CHILD.]

SURGICAL THERAPY OF POSTTONSILLITIC SEPTICEMIA. G. HOFER, Monatschr. f. Ohrenh. **66**:587 (May) 1932.

Hofer thinks that surgical intervention in posttonsillitic septicemia should be undertaken in the earliest stage of propagation of the infection. In all cases in which this propagation is attended by chills, the first real chill should be considered an indication for intervention. The clinical manifestations of posttonsillitic septicemia are variable. Severe chill followed by collapse is the chief general symptom; it is an unequivocal sign of the spread of infectious matter into the blood stream. Locally, a severe tonsillitis is usually followed by abscess formation which forms the basis for a septic condition. The large venous trunks in the neck may be sensitive to pressure, especially in cases of extensive thrombosis, but the pressure sensitivity may be lacking as the disease may take its malignant course without extensive thrombosis of the large blood vessels, especially in the case of primary spread of infectious material into the blood stream. In cases with extensive suppuration of the cellular tissue of the neck, the pressure sensitivity is usually pronounced and early intervention is clearly indicated. The author demonstrates by two case reports that good results can be achieved by ligation of the jugular vein and exclusion of the primary septic focus immediately after onset of the first clinical manifestation of tonsillogenous septicemia, even when no inflammation of the cervical tissue or the sheath of the vessel is demonstrable. In both cases the operation was undertaken within one-half hour of a chill (the first), which had followed soon after draining of an abscess. Collapse of the internal jugular vein in both cases permits the conclusion that there may have been a mycotic venous thrombosis higher up. The author considers location of, and intervention in, the mycotic thromboses not only difficult and dangerous but sometimes impossible. Exclusion of the primary focus, whether still active or not, and interruption of the chief downward route of propagation, the jugular vein, is the first therapeutic requisite, but to be effective it must be undertaken in the first stage of the disease, after the first chill.

EDITOR'S ABSTRACT.

Nose

NEURITIS OF THE NASAL NERVE. FRANÇOIS, Arch. d'opht. **48**:766 (Nov.) 1931.

After first describing a personal observation, the author reviews the anatomy of the ophthalmic branch of the fifth nerve, going into considerable detail in regard to the nasociliary branch. He also describes the composition and distribution of Meckel's ganglion. The symptoms of neuritis of the nasal nerve are oculonasal. 1. There is usually a lesion of the anterior pole of the eye (corneal ulcer with or without hypopyon, iritis, mild cyclitis, mild conjunctivitis, a disturbance of the corneal epithelium discoverable only with the slit-lamp). 2. Very

violent attacks of neuralgic pain occur paroxysmally, out of proportion to the ocular condition. These attacks of pain are accompanied by enormous hydrorrhea. 3. There is marked congestion of the nasal mucosa of the lower turbinate, which is also extremely sensitive to touch. 4. Often there are cutaneous changes, dry herpetiform vesicles or crusts. 5. The pain is promptly relieved by the application of cocaine and epinephrine or butyn and epinephrine to the mucous membrane of the anterior superior part of the nasal fossa, and is unaffected by the instillation of cocaine into the conjunctival sac. 6. Rapid and lasting cure, which was impossible with all the ordinary ocular therapeutic measures, follows this application. The differential diagnosis between the three conditions — neuritis of the nasal nerve, Meckel's ganglion syndrome and ciliary neuralgia — is discussed. The etiology has been ascribed to nasal spurs in two cases by d'Arganaras and d'Espildora, to an acute infectious process by Charlin in two cases and to a mild rhinitis with a deviated septum in the author's case. The onset of this neuritis, according to Charlin, may be due to strangulation of the nerve at its entrance into the nasal cavity by swelling and congestion of the mucous membrane of the nose. The rapid relief obtained by the treatment, used with success in his case, leads the author to support this hypothesis. S. B. MARLOW. [ARCH. OPHTH.]

CICATRICIAL OCCLUSION OF THE CHOANAE NARIUM. V. TORTOLONE, Arch. ital. di otol. **43**:265 (May) 1932.

Tortolone recalls that, in ulcerative or traumatic disorders of the nasal fossae and of the rhinopharynx, a stenosis or occlusion of a cicatricial nature may develop in one or both choanae narium, which stenosis may involve also part of the corresponding nasal fossa and the nasopharynx. While congenital stenosis of the choanae narium is rather frequent, cicatricial stenosis is comparatively rare. The author reports at considerable length the case of a woman, aged 44, of Catania. The examination of the patient as to her general health revealed nothing noteworthy. On examination, by means of anterior rhinoscopy, of the nasal fossae, there were found hypertrophy of the inferior turbinate on the right side and slight hypertrophy on the left side. The hypertrophy of the inferior turbinates was not, however, such as to hamper nasal respiration. Digital exploration of the rhinopharynx revealed that the upper portion of the nasopharynx was obstructed by cicatricial folds of the mucous membrane, which extended from the posterior wall of the pharynx to the choanae narium. A Wassermann test proved positive. The patient was subjected to operation. Under local anesthesia, with long scalpels and with forceps of the Citelli type, an opening was made through the nasal fossae on either side, about 1 cm. in diameter. With the finger the frenula that occupied the rhinopharynx were broken up. Through the two openings thus made two rubber tubes were inserted. This was done with a twofold purpose: to keep the orifices made open and, at the same time, to provoke necrosis by compression of the residual cicatricial tissue. The tubes were always of the same caliber and were changed every two days. Tissue removed was examined histologically and was found to be cicatricial. After a little more than a month of such treatment, the patient was dismissed from the clinic in the best condition and with the nasal fossae entirely permeable. EDITOR'S ABSTRACT.

MEDIAL NASAL FISTULAS. F. R. NAGER, Monatschr. f. Ohrenh. **66**:60 (Jan.) 1932.

Nager reports four cases of medial nasal fistula. Three of the fistulas may be called primary nasal fistulas; the other, as a secondary fistula resulting from rupture of a dermoid cyst following trauma. One of the primary fistulas was located on the membranous septum and was combined with a congenital fistula of the ear. Pathogenesis and therapy are discussed. EDITOR'S ABSTRACT.

SYMPTOM OF ACUTE SUPPURATION OF SPHENOID SINUS NOT PREVIOUSLY DESCRIBED. K. M. MENZEL, Monatschr. f. Ohrenh. **66**:81 (Jan.) 1932.

Menzel briefly discusses the symptomatology of sphenoiditis and reports a symptom observed by him in several clinically demonstrated cases of acute suppuration of the sphenoid sinus. The symptom was lacrimation, and in each case it was one of the outstanding symptoms. Four case reports are given. The author thinks lacrimation is either due to an indirect effect of the inflamed mucous membrane of the sinus, which is innervated by the trigeminus, on the neighboring nerves, especially the sphenopalatine ganglion, or to a direct reflex action due to spread of the inflammation to the sphenopalatine and neighboring nerves. The author calls the attention of his colleagues to the symptom of lacrimation in inflammation of the sphenoid sinus in the hope of finding out whether this symptom is as rare as indicated by study of the literature or whether, if looked for, it will be observed much more frequently.

EDITOR'S ABSTRACT.

FOOD IDIOSYNCRASIES AS OCCASIONAL CAUSE OF VASOMOTOR RHINITIS AND ASTHMA. E. URBACH, Monatschr. f. Ohrenh. **66**:160 (Feb.) 1932.

After reviewing the literature dealing with foods as an allergic cause of asthma and vasomotor rhinitis, Urbach presents an exact method of determining the nutritive allergen and a therapy for these respiratory disturbances due to alimentary idiosyncrasies. The types of asthma and rhinitis referred to by the author are those that occur as the sole reaction to a nutritive allergen, not as one of many symptoms of general anaphylactic shock of nutritional origin. The method of determining the allergic food consists in administration of a food protein preparation diet. Three fourths of an hour before each meal, the patient partakes of the food protein preparation corresponding specificially to the proteins to be included in his meal. If the objective and subjective symptoms of the disease disappear from three to five days later, then one protein preparation after another is omitted until the nasal or asthmatic symptoms reappear. In this way the nutritive allergen, or allergens, can be determined exactly. Therapy consists in consuming the allergizing food twice daily for three or four weeks, always preceded by oral administration of the specific corresponding food protein preparation. The author recommends this method of determining the allergen because it is not subject to error like the subcutaneous method, and is less expensive than the elimination diet method of Rowe. The therapy described is recommended because it is much more rapid than the active desensitization, with gradually increasing oral administration of the nutritive allergen, and above all it is without danger to the patient.

EDITOR'S ABSTRACT.

RADICAL OPERATION ON FRONTAL SINUS ACCORDING TO LOTHROP-SÉBILEAU. V. NASIELL, Monatschr. f. Ohrenh. **66**: 175 (Feb.) 1932.

Nasiell reports a case of frontal sinusitis in which at first conservative, and later more radical, treatment was given, but the condition resisted all intervention until the operation of (Jansen) Lothrop-Sébileau was undertaken. The wound healed completely with a good cosmetic result. This method of operation was named after the two men who conceived the principle of the method independently of each other. The principle of this method is a radical operation of the sinus with production of the widest possible communication between the frontal sinus and with preservation of the external structure of the frontal region. The author briefly describes the steps of the operation and discusses the operation with special reference to some minor difference between the methods of Lothrop and of Sébileau. Lothrop recommends making the trepanation opening only two-thirds inch in diameter; this the author considers too small, preferring an opening large enough to see all possible lesions as recommended by Sébileau. He questions whether removal of the posterior sloping part of the floor of the frontal sinus advised by Sébileau does not make continuous drainage difficult. Contrary to

Lothrop and Sébileau, the author finds no reason why the entire floor of the diseased frontal sinus should not be removed to obtain better access to the cavity and maintain better drainage. The only objection that has been made to this procedure is the connection of a healthy sinus with a diseased one by removal of the septum sinuum frontale in unilateral sinusitis. In two such cases from the author's own experience, this intervention was not attended by any ill results. The author thinks that although the surgical intervention described is extensive and the trend at present is toward conservative treatment of sinusitis, a combination of the frontal sinus operations of Jansen, Lothrop and Sébileau is indicated in severe cases, especially in those in which operation has already been done without success and in which, due to cicatrization, flaps of mucous membrane for lining the connection between the frontal sinus and the cavum nasi are difficult to obtain. It is a radical sinus operation that produces no facial deformities, even in cases of the most extensive changes in the sinus, and as such it is a valuable addition to the surgery of the frontal sinus.

EDITOR'S ABSTRACT.

TREATMENT OF OZENA WITH PANCREATIC FERMENTS. J. REICH, Monatschr. f. Ohrenh. **66**:310 (March) 1932.

Reich reports that the use of pancreatic ferments in ozena constitutes a valuable symptomatic treatment for the support of therapy for ozena. This value is based on the power of the pancreatic ferments to digest the ozenous crusts while leaving healthy tissue unharmed. The pancreatic ferments were used in the form of a salve and a powder.

EDITOR'S ABSTRACT.

GLOSSITIS IN PERNICIOUS ANEMIA. W. MAGER, Monatschr. f. Ohrenh. **66**:315 (March) 1932.

Mager contributes two case reports to the casuistic of glossitis in pernicious anemia and discusses the importance attached to recognition of this condition, which he considers not only an early symptom but frequently an initial symptom of pernicious anemia. The subjective symptoms are intermittently occurring sensations of pain, burning and soreness of the tongue and oral mucous membrane which are exacerbated by ingestion of hot or acidic foods. Objectively, the tongue has a smooth, polished-looking surface, and occasionally the tip edges and dorsal surface appear inflamed. In some cases there is formation of small vesicles and loss of epithelium extending to the soft palate and the buccal mucous membrane, the condition then resembling aphthous stomatitis or even syphilitic stomatitis. The two cases reported were of this type. Diagnosis was determined by the blood picture which revealed hyperchromia, anisocytosis, poikilocytosis, relative lymphocytosis and the presence of megalocytes, although the erythrocytes were not decreased in number and there was no clinical symptom of pernicious anemia except the glossitis. In one case the glossitis existed when the only manifestations of changes in the blood were a slight poikilocytosis and a slight lymphocytosis. The author emphasizes the importance of examining the blood picture in cases of stomatitis to avoid overlooking an existing pernicious anemia, especially in view of the beneficial results of early liver therapy. EDITOR'S ABSTRACT.

NEGATIVE RESULT IN TRIAL IRRIGATION OF MAXILLARY SINUS AND HIGHMOROSCOPY. M. SLOBODNIK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:320 (Feb. 29) 1932.

Slobodnik states that a negative trial irrigation of the maxillary sinus does not guarantee a healthy mucous membrane. Pathologic changes of the mucous membrane may be present in spite of a negative trial irrigation and a negative roentgenogram; this is especially true in cases with polypous or cystic changes due to catarrhal inflammation of the mucous membrane. The presence of minute particles of pus or mucous secretion in the irrigation fluid may indicate disease

of the sinus. If the roentgenogram as well as trial irrigation is negative, direct endoscopy constitutes a reliable method of examination of the mucous membrane of the maxillary sinus. The author reports five cases in which trial irrigations and roentgenograms were negative, but he was able to make the diagnosis of maxillary sinus disease by direct endoscopy. EDITOR'S ABSTRACT.

RHINOGENOUS CEREBRAL ABSCESS OF FRONTAL LOBE COMPLICATED BY GENERALIZED PYEMIA. JUNG, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **22**:145 (Feb.) 1932.

Jung reports a case of rhinogenous abscess of the frontal lobe complicated by generalized pyemia in a boy, aged 12, which was followed by recovery. As there was no clinical basis for assuming a thrombosis of the large sinus, the author thinks the pyemia resulted from invasion of the many veins near the site of operation by infectious agents. EDITOR'S ABSTRACT.

INVESTIGATIONS OF SO-CALLED OZENA SKULL. O. FLEISCHMANN, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **22**:167 (Feb.) 1932.

Fleischmann says that in the clinical picture of ozena the peculiarities of the skull formation are the most unusual manifestations and the most difficult to explain. The author reports his study of this subject, which has led him to the conclusion that the changes in the skull are not the result of ozenous processes nor are they the cause of ozenous processes, but rather the skeletal changes of the skull, and the ozenous processes are both due to the same cause, namely, an ectodermal defect. He thinks the relationship in congenital sweat gland deficiency is undoubtedly the same, and the analogous skull formation in genuine ozena is evidence of the same relation in that disease. EDITOR'S ABSTRACT.

Miscellaneous

SPEECH DEFECTS IN CHILDREN. LEOPOLD STEIN and I. J. WOLF, Arch. Pediat. **49**:27 (Jan.) 1932.

1. The following speech and voice disturbances are reviewed: hearing, dumbness, partial and universal stammering, organic peripheral dyslalia, dysarthria, stuttering, rhinolalia, hoarseness and falsetto voice.

2. The problem of speech disturbances is a medical one, as has been stressed by others. A knowledge of medicine is necessary for the diagnosis and treatment of most of these conditions.

3. Stammering and stuttering must be differentiated. The former is a disturbance of the articulation; the latter, a speech hesitating in nature, is dependent on psychic states. Stages of development from early childhood are evident.

4. Organic causes of stammering must always be ruled out because of their importance in prognosis. Common among these are mental backwardness, birth trauma and poor hearing. WADDELL, University, Va. [AM. J. DIS. CHILD.]

POSTOPERATIVE CARE OF FOREIGN BODY IN THE LUNG IN INFANTS AND YOUNG CHILDREN. M. F. ARBUCKLE, South. M. J. **25**:456 (May) 1932.

Young children are more liable than older children to have complications, such as shock, cyanosis, dyspnea and sepsis. In robust infants with no complicating condition, ordinary examination and routine bronchoscopy are indicated. In the case of an infant who enters the hospital in a state of shock, with cyanosis and dyspnea, immediate supportive treatment is imperative. A tracheotomy set should be available, and the surgeon should be in constant attendance pending the completion of arrangements for bronchoscopy. A routine bronchoscopy may then be done with safety.

The patient must be placed in an oxygen tent and watched for subsequent developments. If signs of sepsis develop, drainage should be provided by a tracheotomy and the frequent application of suction. The author considers this the surest method of avoiding serious complication in the lungs.

The infant who has had a foreign body in the lung for a considerable period and who is septic as the result of infection in the lung will show great exacerbation of all symptoms after the operation. Tracheotomy with lower bronchoscopy is the operation of choice in such cases.

These patients should always be in an oxygen tent and should receive a great deal of fluid and have excellent postoperative care. The author prefers expectorant mixtures to the use of morphine or atropine in cases showing extensive secretion from the bronchi.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

INJURY TO THE BULB OF THE JUGULAR DURING PARACENTESIS. P. MANGABEIRA-ALBERNAZ, Ann. d'oto-laryng., Feb. 1932, p. 151.

The author calls attention to the anatomic relation between the bulb of the jugular and the floor of the cavum tympani and how in a well developed bulb the form and structure of the floor of the tympanic cavity show marked modifications. The bony covering of the bulb may be reduced to a thin transparent lamella. In some cases the mucous membrane and vascular layer are in contact with each other. This relation explains the primary phlebitis occurring in an acute otitis media.

The relation of the jugular bulb in these cases to the posterior inferior quadrant of the membrana tympani places it in the area of predilection for paracentesis, and in these cases it is practically impossible not to strike the bulb.

The author reports the case of a child, 9 months old, in whom this accident occurred. The case was one of bilateral acute otitis. The condition called for paracentesis, which was done without anesthesia. Nothing abnormal was noted. However, two hours later hemorrhage from the external canal occurred, brought on during a crying spell. The bleeding was copious and was controlled with difficulty by packing the external canal, which packing had to be renewed and left in place for forty-eight hours. In a month's time the child had recovered from the bilateral otitis. Two weeks later, without any evidence of otitis media, meningitis developed, and the child died.

The author does not believe that the meningitis was related in any way etiologically to the bleeding from the bulb during the previous otitis media. He points out the extreme rarity of this accident. In the literature from 1922 to 1930, only one case was reported. In 1908, Koerner had collected five cases. Schwartze appears to have been the first to have recorded this phenomenon, and eighteen cases appear to have been observed in all.

The accidental puncture of the bulb of the jugular is generally without complications. However, occasionally septic pyemia has occurred.

Trautmann points out that three conditions are indispensable in order to diagnose the presence of a jugular bulb in the tympanic cavity. First, is the presence of a bluish area in the posterior segment; second, the sharp outlines of the upper limits of this area; third, modification of the light reflex after compression of the jugular vein in the neck. As a matter of fact, dehiscences in the inferior wall of the tympanic cavity are rare. Koerner found defects in only 30 of 449 skulls examined. They were more frequent on the right side. The reason the bulb of the jugular sometimes bulges into the tympanic cavity seems to depend exclusively on the size and degree of curvature of the sigmoid sinus. When the sigmoid sinus takes a nearly straight course till it reaches the jugular, the bulb then is very little developed; when the sinus is somewhat anterior and much curved, the bulb enlarges to such an extent as to press the floor of the tympanic cavity and the cochlea upward, giving rise to the dehiscence.

In conclusion, he points out that the bulb of the jugular vein is sometimes markedly developed and destroys the inferior floor of the tympanic cavity, thereby coming into direct relation with the mucous membrane. In such cases the vessel

almost surely is damaged during paracentesis causing a profuse hemorrhage. The accident is inevitable as there is no sure way of seeing the immediate relations between the vessel and the mucous lining of the cavity but it occurs rarely. The entire literature in more than sixty years mentions only nineteen cases. The hemorrhage can almost always be controlled by means of tight packing.

P. VAN DER LEEK, Stockton, Calif.

DIFFERENTIAL DIAGNOSIS OF OTOGENOUS ABSCESSES OF TEMPORAL LOBE. H. BRUNNER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:136 (April 25) 1932.

Brunner says that one of the reasons for the difficulty of diagnosing otogenous abscesses of the temporal lobe is that inflammatory diseases of the cerebrum not of otogenous origin may present the symptoms of otogenous cerebral complications. He reports several cases of this type, discussing in detail the diagnostic difficulties presented by each one. In case 1 the cerebral disturbance appeared with the symptoms of otogenous encephalitis of the frontal and temporal lobe, but was later diagnosed as acute influenzal encephalitis occurring with a left lateral chronic otitis. Case 2 concerns a tuberculous patient with infection of the middle ear who presented cerebral symptoms. Autopsy, however, indicated that the tuberculoma found in the left cerebral hemisphere did not originate from the ear but was due to spread of the miliary process from the lungs. In case 3, clinical examination revealed disease of the right cerebral hemisphere. An exacerbation of a chronic right lateral otitis media, together with rapidly increasing cerebral symptoms, pointed toward an otogenous brain abscess. At autopsy a large circumscribed abscess was discovered in the medullary layer of the right gyrus centralis which had no connection with the petrous bone. The abscess was attributed to metastases from a suppurative pulmonary infection already healed at the time of examination; this was possible because, according to the anamnesis, the beginning of the cerebral symptoms dated far back. Case 4 concerns a child, aged 4 years, who exhibited symptoms of a cerebral abscess about two months after apparent healing of otitis media that developed during scarlet fever. The localization of the abscess could not be determined at first, and the otogenous nature of this complication was contradicted by the results of examination of the ear and by roentgenograms. It was only on injection of air into the ventricles that the existence of an obstruction in the right cerebral hemisphere could be definitely established. Operation revealed a large abscess in the right temporal lobe, drainage of which resulted in recovery. A definite conclusion as to whether the abscess was metastatic or otogenous cannot be made.

EDITOR'S ABSTRACT.

Society Transactions

AMERICAN OTOLOGICAL SOCIETY

ROBERT SONNENSCHEIN, M.D., *Reporter*

Sixty-Fifth Annual Meeting, May 17-18, 1932, Atlantic City, N. J.

L. W. DEAN, M.D., *President, in the Chair*

Tuesday Morning Session, May 17, 1932

CYTOLOGIC AND BACTERIOLOGIC STUDIES OF OTITIC DISEASE: PRESIDENTIAL ADDRESS. DR. L. W. DEAN, St. Louis.

During the last year, we have interested ourselves in various fundamental problems of otolaryngology. The viscosity, the p_H, the nitrogen content and the carbon dioxide content of the secretions have been studied. By interfering with calcium metabolism we have produced an experimental eosinophilia followed by infection of the membrane. The influence of acidosis and alkalosis on the eosinophilia has been studied. Secretory nerves have been stimulated to note the effect on the mucosa; also the influence of various drugs has been observed. We have produced an experimental allergy. The work on the physics, the anatomy and the pathology of the ear has been continued.

These problems are complicated, and the results secured have more influence on nasal sinus disease than on disease of the ear. Practical results came from the cytology of the discharge.

In our work on the bacteriology of the ear, we have not concerned ourselves so much with the routine study of all cases, but rather have made complete studies of groups of interesting cases.

The hemolytic streptococcus and the hemolytic staphylococcus have been the outstanding etiologic factors in the cases of acute otitis. The importance of securing the material for bacteriologic examination at the time of myringotomy and at the time of operation on the mastoid has been repeatedly emphasized.

Pneumococcus, type III, has often been found at the time of myringotomy. It has been absent on repeated examination of the discharge from the ear and was then found again at the time of operation on the mastoid.

We have found the normal tympanic cavity to be sterile as a rule, and if cultures are made at the time of myringotomy, the etiologic factor will probably be discovered. Following myringotomy there is an invasion of contaminating organisms, and it is almost impossible to say what the significance of an organism is if one is found. In many of our cases the true etiologic factor has been lost in this mass of contaminants.

All of the cases studied were examined for both aerobic and anaerobic organisms. Most anaerobic organisms are secondary invaders; they have much to do with the production of the foul odor in the ear. The foul odor is due to the presence of incompletely oxidized cleavage products. These organisms are found to be more abundant in destructive processes. Ordinarily they are harmless, but they may contribute to a pathologic process under suitable conditions. They cannot be disregarded as important factors. For instance, Bacterium melaninogenicum was found in one case of mastoiditis with thrombophlebitis, abscess in the neck, septicemia and finally meningitis, and it was the only organism found in the neck, in the blood stream and in the spinal fluid.

The pathologic process so changes resistance that Bact. melaninogenicum becomes an etiologic factor. This process is not so different from that in cases reported by Shurly, in which deficient diets resulted in the nonpathogenic organisms becoming pathologic.

The formation of serous discharge in early otitis is the first defense against infection. It is the result of irritation, and the excess of fluid is to get rid of the irritant. At first this secretion has a mild bactericidal action. Some time ago, Mr. Linton showed that this action is soon lost. However, this current of secretion plus its bactericidal action sometimes overcomes the invasion by the bacteria.

In the event the irritant is not washed away, but reaches the cell, I am inclined to think that the next defense is the desquamation of groups of superficial epithelial cells. These cells are thrown off in small masses, carrying the bacteria with them.

Pneumococcus, type III, has continued to be a dangerous organism. We have found it often at the time of mastoidectomy when its presence was not suspected owing to the fact that it could not be picked up in the discharge from the ear. These cases have all behaved in the same way as routine infections of the mastoid, and the finding of the organism at operation does not in any way influence the prognosis. The finding of this organism at the time of myringotomy has a decided bearing on the prognosis. In every case in which this organism has been found it has been necessary sooner or later to perform an operation on the mastoid. One should exercise the best clinical judgment.

Many years ago I investigated a large series of cases of chronic otorrhea in patients with pulmonary tuberculosis and was unable to find evidence in the tissue or bacteriologic evidence of tuberculosis of the ear in all of them. In the proved cases of tuberculosis in my experience there was a poor result whether or not operations were performed. The infection is through the blood stream, and the pathology is that of tuberculosis of the bone.

In a case of maxillary sinusitis, we secured Bacillus tuberculosis from the chronic discharge from the sinus, and the injection of this discharge into guinea-pigs gave positive results. The sinus was later operated on, and the opinion of the pathologist was that there was no indication of tuberculosis of the lining of this chronically infected sinus.

When a person with a tuberculous lesion develops an infection in any part of the body, B. tuberculosis is likely to invade the tissue as a contaminant, and the finding of the organism does not necessarily indicate that the lesion is tuberculous.

In connection with the cytologic examination of the discharge from the ear, in one case yeast cells were found in the discharge in great abundance. The patient was an infant suffering from malnutrition. Torulae are found in the throats of poorly nourished infants. They may occur in the external auditory canal in any case. When present in the throats of infants or in the otitic canals, they have little or no clinical significance. The infection of the ear in the case mentioned was not clinically coccidiosis. The condition of the ear cleared up in a few days. Yeast cells were undoubtedly contaminants and had no significance.

In making cytologic studies of otitic discharges, we became interested in the frequency with which cholesterol crystals plus fatty degenerated cells were found. These two things were present many times more frequently than ever before. The reason for this is that in studying the cells in the washings from the ears the material was centrifugated and the precipitate examined. The cholesterol crystals were found in the precipitate.

Cholesteatoma is said to be definitely diagnosed by the presence of cholesterol crystals plus fatty degenerated cells in the discharge from the ear. Some authors think that the presence of cholesterol crystals alone is pathognomic of cholesteatoma (Rejtö, Alexander: Cholesteatoma, ARCH. OTOLARYNG. **13**:709 [May] 1931).

We have found the crystals plus the fatty degenerated epithelial cells in so many cases that we are inclined to doubt the significance of the findings. Cer-

tainly, clinically one would not advise radical operation on some of the patients who had crystals in the otitic discharge. The crystals of cholesterol were present in the discharges in a much larger percentage of cases of chronic otorrhea than the number of cases of cholesteatoma found when operation was performed. The fact that cholesterol and fatty degenerated epithelial cells are found in the discharge in the external auditory canal does not necessarily mean that the cholesterol and cells are coming from the middle ear.

It seemed advisable to ascertain whether the cholesterol in the otitic discharge was peculiar to cholesteatoma. With the acetic anhydride test or the sulphuric acid test, together with the polariscope, certain studies were made.

In cerumen there was found by the colorimetric test a high percentage of cholesterol. The color reaction was more marked than we have secured from any discharge. The cerumen was densely impregnated with crystals. These crystals do not have the characteristic form of cholesterol. They are, however, probably cholesterol. The uncertainty is sufficient to cast doubt on the significance of cholesterol crystals in any discharge found in the external auditory canal. Cholesterol was found in the otitic discharge in cases of acute otitis. The older the discharge the greater is the amount, as a rule. It was quite evident that the presence of cholesterol was not peculiar to cholesteatoma.

After we had begun our work, we found that Rejtö had made a study of the colorimetric method of determining the amount of cholesterol. He thought that a strong color reaction was diagnostic of cholesteatoma and that a weak reaction excluded it. Salkowski, according to Rejtö, is of the opinion that the cholesterol in cholesteatoma is not always crystalline. He estimated that there is from 18 to 23 per cent of cholesterol in cholesteatoma, and 10.6 per cent in the normal epidermis. Lautenschlager believes that the cholesterol in cholesteatoma causes absorption of the bone. With the polariscope, crystals can be found in the purulent discharge of recent cases of acute otitis. While they are not the characteristic cholesterol crystals, they do not differ from the crystals sometimes found in known cases of cholesteatoma. However, little is known of the uses of cholesterol. It is a protective agent. It combines with toxic substances.

In sections of temporal bone with mastoiditis we often found diamond-shaped spaces in granulation tissue which contained cholesterol before it was dissolved out by the solutions used. There is no suggestion of cholesteatoma in or near these masses, so that in chronic suppurative otitis, cholesterol in the ear is certainly not peculiar to cholesteatoma. As yet we have not determined the percentage of cholesterol in granulation tissue in the mastoid.

Because of the clinical significance of cholesteatoma and the diagnostic significance of cholesterol in the discharge from the ear, it seemed desirable to determine just what amount of cholesterol was present in the discharge. Cholesterol is insoluble in water. Even if some substance such as carbon tetrachloride, in which it is soluble, is used, the location of the cholesteatoma or cholesterol in granulation tissue would determine the contact which the drug would have with the cholesterol. It does not seem that the same opportunity for the cholesterol to get into the otitic discharge would always be present.

Colorimetric determinations are made as follows: The ear washings are evaporated to dryness over a water bath; the residue is saponified with 3 per cent potassium hydroxide in 95 per cent alcohol for from twenty to thirty minutes (over a water bath). The cholesterol is extracted from the alcohol with petroleum ether in a separatory funnel, the layers being separated. The petroleum ether is evaporated, and the residue is extracted with chloroform. Standard cholesterol solutions of different strengths are prepared in order to have one containing approximately the same amount of cholesterol as the unknown. The unknown is made up to exactly 10 cc. with chloroform. Ten cubic centimeters of each of the known standards is pipetted into dry test tubes. Two cubic centimeters of acetic anhydride plus 0.2 cc. of concentrated sulphuric acid is added to each tube (known and unknown), and all of the tubes are subjected to the same light. The color is allowed to develop for about fifteen minutes. By comparing

the unknown with the known closest to the color of the unknown in the colorimeter, the amount of cholesterol in the sample is determined.

Certain factors influence this test. Time, temperature and light influence the color. The method is simple and under uniform conditions gives accurate results. In every case examined crystals of some kind have been found by using the polariscope. Every discharge from the middle ear has given a colorimetric reaction of cholesterol. The subject which is being investigated is complicated, and it will be a long time before we, at least, shall be able to express a definite opinion as to the actual significance of the presence or the absence of cholesterol crystals as a diagnostic factor in cholesteatoma and the significance of the presence of large amounts of cholesterol as a diagnostic point.

We believe that the presence in the otitic discharge of cholesterol crystals with fatty degenerated epithelial cells does not positively indicate the presence of cholesteatoma; neither is it indicated by a marked color reaction for cholesterol.

REPORT OF THE COMMITTEE ON THE STUDY OF PROGRESSIVE DEAFNESS. DR. ARTHUR B. DUEL, New York.

Dr. Duel said that the economic depression had influenced the income of the research fund. He mentioned the allocation of funds and stated that Dr. Wilson would present details regarding the Committee's report.

DR. JOHN GORDON WILSON, Chicago: The report of your Committee on Progressive Deafness this year, is chiefly confined to the work of Dr. Bast, of Dr. Crockett and of Dr. Davenport. It is to be noted that satisfactory investigations are under way at McGill University with Dr. Tait, at Illinois University with Dr. Culler and at Northwestern University with Dr. Anson. Next year we expect to give a full account of this work.

DEVELOPMENT OF THE PETROUS TEMPORAL BONE. DR. T. H. BAST, Madison, Wis.

To date we have serial sections of a representative chronological series of fifty human fetal ears.

We have collected seven ears from young children, some of which are ready for sectioning.

We have constructed fourteen different models of human ears showing growth of the capsule and blood supply.

The following papers on the ear have been published: (1) The Utriculo-Endolymphatic Valve (*Anat. Rec.* **40**:61, 1928); (2) Ossification of the Labyrinthine Capsule (*Laryngoscope* **38**:665, 1928); (3) Osteogenesis of the Human Periotic Capsule (ARCH. OTOLARYNG. **10**:459 [Nov.] 1929); (4) A Comparative Study of the Utriculo-Endolymphatic Valve in Some Common Animals, 1930; (5) Ossification of the Otic Capsule in Human Fetuses (*Contrib. Embryol.* [no. 121] **21**:53, 1930) and (6) Blood Supply of the Otic Capsule of a 150-Mm. (C.R.) Human Fetus (*Anat. Rec.* **48**:141, 1931). A seventh paper, "Development of the Otic Capsule: I. Resorption of the Cartilage in the Canal Portion of the Otitic Capsule in Human Fetuses and Its Relation to the Growth of the Semicircular Canals" (with nine plates and thirty-four figures), has been accepted for publication in the ARCHIVES.

It is now possible to come to some tentative conclusions regarding the question whether the otic capsule is predisposed to subsequent change. In the metamorphosis of cartilage into bone the normal tendency in most bones is completely to replace the cartilage by a primary endochondrial (*geflechtartig*) bone and this by a secondary or lamellar bone, and sometimes the change is directly from cartilage to a secondary bone. Primary endochondrial (*geflechtartig*) bone is always present in bone and represents a stage in bone formation. It is normally removed by vascular buds and, replaced by lamellar bone around the vessels, except for small remnants, which remain between the lamellar bone, the so-called interstitial bone. In the otic capsule, however, the bone formation is incomplete. In the first place, in small isolated areas which present individual variation in number and distribution, the cartilage matrix is not completely removed; only the

lacuna of the cartilage is changed into primary bone, while the cartilage matrix persists in calcified cartilage. Such areas are known as interglobular spaces, cartilage islands or intrachondrial bone. Between these areas an endochondrial (cartilage) bone is formed ranging from a primary (*geflechtartig*) bone to a secondary lamellar bone.

INFLUENCE OF BODY CALCIUM ON OTOSCLEROSIS. DR. E. A. CROCKETT, Boston.

The influence of increased body calcium on the etiology and on the progressive nature of otosclerosis has been much in dispute. For a study of this subject, a grant was given in 1927 to Drs. Crockett and Aub of Boston. It was arranged that the early and more intensive part of the investigation should be carried on in the metabolism wards of the Massachusetts General Hospital, and that later, when the patients returned to their homes, they should still remain under observation and guidance. This plan has been successfuly carried out. The investigation, therefore, has been in progress for nearly four years. Twenty-three cases have been studied. In 1927, for one month, the patients were strictly confined to the special metabolism ward under Dr. Aub's supervision on a carefully prepared low calcium diet, with a special dietitian. The intake of food was carefully weighed and was adequate in everything except calcium. The output of calcium in the body secretions was carefully measured.

The following conclusions were reached: 1. No marked improvement in hearing can be expected in any case in which the diseased process has existed much over a year. On the other hand, where the otosclerosis has not existed over a year and when the amount of change in the bone as judged by the hearing test is not great, an improvement may be effected. In three cases, none over twelve months' duration, the improvement in the hearing was marked; the hearing had become normal (audiometer reading), though the tinnitus had not quite disappeared. This improvement has been held to date, now two and a half years.

2. A slowing of the progress, a possible cessation of the disease, may be looked for in older patients in whom the process is of longer duration after the use of the diet with a diminished calcium intake of the type previously mentioned. Such a diet is easy for any patient to carry out.

3. While the use of parathormone to pull calcium from the body greatly assists in the output of calcium, its use has been found unnecessary. It is expensive and, in order to be administered, necessitates hospitalization. The same result can be obtained in a simpler, slower way by the administration by mouth of from 50 to 60 grains (3.25 to 3.9 Gm.) of ammonium chloride once a day, according to the patient's tolerance.

GENETICAL STUDY OF OTOSCLEROSIS. DR. CHARLES B. DAVENPORT, Cold Spring Harbor, New York.

The study of the influence of heredity on otosclerosis was undertaken by Dr. Davenport of the Carnegie Institute of Genetics, Cold Spring Harbor, Long Island, N. Y. Dr. Davenport directed his investigations to an analysis of the factors responsible for the development of this defect, particularly the genetic factors, using especially the method of mendelian analysis.

He came to the following conclusions: The hypothesis of two dominant genes is not negated in any of the groups of matings. The hypothesis that otosclerosis depends on a single dominant factor, on a single recessive factor, on a sex-linked recessive and a dominant autosomal gene, on two recessive genes (one sex-linked) and on a dominant recessive and a recessive autosomal gene meets with difficulties in application. The conclusion is reached that the simplest formula that approaches agreement with the findings is that of two dominant genes, one of which is sex-linked.

No geneticist believes that the genes which shape the construction and constitution of the body constitute the whole story of the transmission of a morbid condition or a malformation. Characteristics are not transmitted as such from

parent to offspring. What are inherited are hereditary units which interact and give the plant or animal the power to respond in a certain way to a given kind of environment. The genes determine the possibilities; the actual realization of any possibility is dependent also on environment and on hereditary factors. Environment has more influence on the results when acting on a suitable person. The fact that a smaller percentage of otosclerosis occurs in some families than in others appears to show that influences other than the gene are at work.

Is All Hearing Cochlear? Dr. John Tait, Professor at McGill University, Montreal, Canada (by invitation).

The eighth cranial nerve subserves both an exteroceptive (auditory) and a proprioceptive (equilibrium) function. Its equilibrial receptors, anteriorly placed, are the semicircular canals and the utricular macula. The cochlea, posteriorly situated, is auditory. The function of the saccular macula, which lies between the proprioceptive and the exteroceptive sense organs, is unknown. Dr. Tait marshalled evidence in support of the view that it is auditory.

While the cochlea and its accessory apparatus, all of light construction, vibrate in an otherwise immobile head, the saccular macula presumably responds to actual vibration of the head. Some of the otolithic receptors of aquatic invertebrates respond to water-conveyed tremor. In lower vertebrates there are at least two otolithic receptors in the posterior part of the labyrinth, namely, the lagena and the saccular macula. G. H. Parker has obtained evidence that the saccular macula of a fish is auditory. Mr. D. A. Ross, a worker in Dr. Tait's laboratory, has shown that both the lagena and the saccular macula of certain fishes are auditory.

Strangely enough, the croaking mechanism of frogs is designed not primarily to transmit air-borne sound, but for the purpose of causing a tremor of the water, guided by which the sexes are brought together. Some fishes similarly use their air bladder for sexual signaling under water. The voice of vertebrates took origin at an early stage, and then for sexual purposes, it began in relation to the air bladder of fishes. This air bladder became transformed into the lungs of terrestrial descendants. By the transformation the tremor-emitting characteristics of the original air bladder were not sacrificed, but, being effective also for production of air-borne sound, were retained as the basis of voice in all the higher vertebrates.

Dr. Tait holds that it is especially during voice production that the saccular macula is stimulated. It is thus a proprioceptive and not necessarily an exteroceptive auditory organ. It responds to the tremor of the head that inevitably accompanies spoken voice. Only when one whispers can one hear one's own voice by cochlea alone. An R sound cannot be whispered, because it involves a vibratory trill. It is because of the absence of saccular stimulation that one's own voice, as reproduced by phonograph, fails to be recognized.

DISCUSSION

Dr. J. Gordon Wilson, Chicago: The saccula has gone through many vicissitudes; one point is clear, that is, the rôle it has been supposed to play in equilibrium will have to be either abandoned or greatly modified. All evidence at present points to its importance as a receptive mechanism for what may be called gross vibrations, an interpretation which Dr. Parker suggested some years ago as a result of his experiments on fish. Dr. Tait has confirmed Dr. Parker's results in the frog and has amplified them in great detail. The clinician thinks back on those puzzling cases in which, with good evidence that the cochlea was destroyed, the patient seemed to pick up vibrations with extraordinary ease.

In his discussion of the relation of the larynx to the development of the lung, Dr. Tait raises important problems: How far was the lung developed as an adjunct to the larynx and emotional reactions? How far was the larynx developed in a position where vibrations could be easily produced? While it appears that the mass of evidence supports the latter hypothesis, it may well be that both have to be taken into account.

MR. R. L. WEGEL, New York: Professor Tait has reported an interesting demonstration which indicates that in lower animals the sacculus and utriculus are sensitive to vibration, and that their excitation constitutes all or part of the "hearing" in such animals. The thought suggests itself that in these animals the threshold referred to as "feeling" in man is lower than that of hearing, if indeed hearing as in higher animals exists at all. There are at least two measurable thresholds in the human ear, auditory and "feeling," and at least six possible thresholds. It is to be taken for granted that any one of the nerve terminations in the labyrinth—the ampullae, sacculus, utriculus and organ of Corti—may be excited by sound vibrations if they are loud or violent enough. Some observers describe a sensation of dizziness at the "feeling" threshold, suggesting excitation of one or other of the semicircular canals. The sacculus and utriculus appear to be more favorably situated for picking up vibrations, which circumstance gives color to the hypothesis that they are responsible for the hearing threshold at low frequencies. There is against this the fact that the hearing threshold is continuous from the highest to the lowest perceptible frequency, and the high frequencies are without doubt perceived in the cochlea. Professor Tait's paper is valuable not only in the discussion of the physiology of lower animals, but also in bringing attention to the likelihood that the sacculus and utriculus, as well as the cochlea, are excited at low frequencies in man.

DR. EUGENE R. LEWIS, Los Angeles: As Mr. Wegel was talking I recalled a patient who complained of a peculiar nausea every evening, at the same time, during the summer. It was not associated with vomiting or pallor. At this hour, bats flew from the nearby church belfry. The bat's squeak is a supertonic tone, and this may have irritated the chorda tympani, causing not only nausea, but sialosis.

MR. R. L. WEGEL, New York: By increasing the intensity of sound one can arrive at a point where people complain of feeling dizzy or are nauseated. This suggests that the vibrations affect the semicircular canals. As the saccular macula is more favorably situated, it has received these vibrations, which brings up the interesting point of hearing by means other than the cochlea.

DR. STACY R. GUILD, Baltimore: These views are more or less historical. Corit, Henson and Helmholtz, among others, thought that the cochlea served only for analysis of tones, and that the maculae and cristae served to mediate other types of vibration. Henson and Lucae, in the first decade of this century, clung to the view that the vestibular end-organs also have an auditory function. So far laboratory examinations of the macula sacculi in cases of total deafness have not given anything of interest, but with Dr. Tait's work in mind, I am going to give the problem more attention, especially in cases in which a discrepancy exists between the amount of the loss of hearing and the microscopic pathologic changes in the middle and inner ear.

DR. EDMUND P. FOWLER, New York: The old theory was that the cochlea was for the differentiation of sound, and the other structures detected noise. Dr. Tait's work seems to be proving this. Perhaps the irritation or stimulation of the saccule disturbs the cochlear function, just as any noise will mask any other noise, or as tinnitus interferes with hearing. This research suggests that tinnitus caused by noise comes from inflammation of the saccule, and not from the cochlea.

DR. LORENTE DE NO, St. Louis: Years ago I made some experiments on ablation of the saccular macula in the rabbit. I found no spontaneous symptoms. The labyrinthine reflexes of the eyes seemed to be quite normal, but when the rabbit was turned on the turning table and the ocular reflexes were recorded, there appeared, at the same time as the nystagmus, slow changes in the tonus of the ocular muscles, which, of course, represented slow oscillations of the eyes. I do not regard these experiments as conclusive, because in order to destroy the saccular macula it is necessary to open the membranous labyrinth, and the perilymph and endolymph have a quite different pressure after the operation. It may be said, then, that until now there has been no physiologic evidence of any participation of the

saccular macula in the regulation of the equilibrium and allied function. However, anatomically, the evidence is strongly in favor of the view that the saccular macula must have a function similar to that of the macula of the utricle and quite different from that of the cochlea. In histologic preparations stained by the silver methods, I have been able to follow the nerves of the saccular maculae into Scarpa's ganglion and thence into the medulla, where the endings are partly in common with the nerves of the utricular macule. On the other hand, the cochlear nerve had nothing in common with the saccular nerves. One must also think that the saccular macula has a function related to that of the utricle and semicircular canals and independent of that of the cochlea, but, of course, nothing can be stated until physiologic evidence is available.

Dr. John R. Page, New York: I reported a case of paracoustic vertigo some years ago in which sound produced a movement of the eyes and a tendency to fall in the plane of the right anterior vertical canal. The hypersensitiveness in this canal became so disturbing that the patient could not speak above a whisper without falling. Finally, relief was afforded by an operation on the labyrinth. The vestibular and cochlear regions were opened, and the anterior vertical and horizontal semicircular canals were removed.

Dr. John Tait, Montreal, Canada: Dr. J. S. Fraser once told me that the older physiologists associated the saccule with the cochlea. In lower vertebrates, a number of different receptors occur in this auditory part of the labyrinth. One must keep in mind the distinction between response to noise and reception of tone. Some of the primitive auditory receptors may be for noise, and others for tone. In answer to Dr. de No, stimulation even by the utricular otolith is not constant. The muscles of even a delabyrinthized animal can hold posture. When, by a tilt of the head, the utricular otolith slips, the utricle changes a set posture. Both the utricle and the saccule may be somewhat analogous in their mode of stimulation. Dr. Wilson has found a valve between the membranous saccule and the membranous utricle in mammals. To discriminate between the different auditory receptors in the frog, we shall have to examine the animal in its natural surroundings in the springtime. We shall then combine laboratory with field experiments.

Physical Measurements and the Physiology of Excitation of the Auditory Nerve. Mr. R. L. Wegel, New York.

Practically all phenomena of hearing studied by so-called physical measurements involve, as an essential component, certain reactive properties of tissue. In order to discover the details of the physical mechanism from information so obtained, it is therefore necessary to know the properties of the tissue of the nerve tract as a meter of stimulus, or in the absence of such knowledge to be content with the best obtainable approximations. This paper deals with the interpretation of certain data on excitation of tissue into a form applicable, as nearly as possible, to the problem of hearing.

The diffusion theory of excitation of Nernst provides a quantitative method of calculation of excitation occurring within a time interval of three chronaxia. For longer times the incidence of changed metabolic rate modifies the laws. The latent additional data of Chauchard indicate an increase in excitability by a factor of 10 or more. The indications of discrimination data are that the auditory nerve tissue responds in this supernormal phase near the threshold.

Absolute pressure acuity at low frequencies varies inversely as the cube of the frequency, indicating that the end-organ here is responsive at the threshold to its mechanical acceleration. Stated in other words, the velocity of motion required by the basilar membrane for threshold excitation is proportional to the time interval between successive impulses in the sound wave when this interval is long compared with the chronaxia of the nerve.

An important factor in binaural hearing is the cross-connection between the two sides of the auditory nerve tract. From observations on binaural beats by Stewart,

it is concluded that the time required for this cross-propagation is from 0.010 to 0.001 second, depending on the frequency, in the normal ear. The phenomenon disappears as the frequency is increased, at about 1,000 cycles, presumably because of the statistical nature of the generation of nerve impulse, resulting in part from the encroachment of the stimulus on the refractory phase of the tissue.

DISCUSSION

PROF. K. S. COLE, New York: Mr. Wegel has emphasized the complete chain of mechanism involved in hearing. His data suggest that in the range below 1,000 cycles, the mechanical system from the outer ear and the nervous system to the brain are both capable of transmitting subthreshold intensities, but the sensory end-organ is the weakest link in the chain. As shown by Adrian, the problem of the sensory end-organ is an outstanding physiologic one at this time.

If if is assumed that acceleration of a hair will bring the sensory cell into activity, then at frequencies above 1,000 it may be stimulated without impulses reaching the brain, since another mechanism, the nerve itself, is responsible for the behavior of the whole. The nerve, however, is a problem by itself.

The work of Galvani, in the eighteenth century, was the forerunner of the birth of physical chemistry. Recently, Nernst gave to physiology his theory of nerve stimulation. Mr. Wegel's work is both stimulating and instructive. It suggests that the most important problems of hearing are those physiologic problems which are being powerfully attacked at the present time—the sensory end-organs and nerves.

DR. JOHN TAIT, Montreal, Canada: Mr. Wegel has utilized the latest studies in work on nerve conduction, and the way in which he has done this shows what a good physiologist is hidden in the Bell Telephone Company. He takes the subjective sensory findings as determined in his own laboratory, and attemps to explain them in the light of the most recent physiologic data. He employs mathematical analysis to explain the happenings within the cochlea. This side of his paper I am incompetent to discuss, and I think it is interesting that papers of this type are presented here.

DR. ELLISON ROSS, Chicago: The transmission of the nerve impulse is a common physiologic process, comprising metabolism, oxidation and reduction, elimination of energy, fatigue and recovery. Another factor, however, that is disturbing enters into the problem, that is, electricity. A nerve trunk is a bundle of fibers more or less electrically insulated from each other. The general plan of the nervous system is that of a common commercial electrical system. The Wever-Bray phenomenon, with the auditory nerve, argue strongly for the importance of electricity in stimulation of the nerves. Electricity is the great problem still ahead. Mr. Wegel has employed highly mathematical and physical methods of attack which will constitute a great advance in this field. However, we must not draw too general conclusions. Living tissue, chemical composition, temperature, blood supply, toxins and the colloidal state have their influence. Small changes in any of these factors may make great differences in the composite result in a nerve.

DR. EUGENE R. LEWIS, Los Angeles: One factor that seems worthy of consideration is inertia. The initiation of activity, whether in one cell or in a combination of cells, may require more force than is required in the maintenance of activity. Ultimately, increasingly intense stimuli are followed by fatigue or exhaustion of the cell. In some types of nerve mechanism there seems to be a self-contained factor which makes for continuation of action until cessation or fatigue occurs. As the store of available energy for response approaches the exhaustion point, the cell shows physical changes in form and size. A final stage, cellular dissolution, is reached when opportunity for restoration is denied the cell. One sees evidence of maximal, minimal and pathologic degrees of response.

PROF. H. DAVIS, Boston: The slowly acting tissues, such as the smooth muscle tissue of the alimentary canal and of the blood vessels, can be thought of as the

opposite extreme from the ear. I conceive of the ear as having extremely rapid action. The impulses that follow the auditory pathways have frequencies up to 900 vibrations per second. It is in this region that a discontinuity occurs, and I suspect that there is significance in that coincidence.

The activity of the sense organ is divided into the fast and slow types. The slow organs are stimulated by continuous excitation, such as tension or temperature. In the tactile cells, where the discharge is confined to the moment of contact, there is rapid adaptation of the sense organ to the stimulus. The cochlea, as Dr. Tait suggested, is a modified tactile organ. When the conditions are favorable for the transmission of sound waves to the auditory nerve, it is possible for the impulse to follow at fairly high frequencies, the limit being imposed by the recovery of the nerve itself.

Mr. R. L. Wegel, New York: In regard to the discussion by Professors Cole, Tait and Ross, I wish to express my gratitude to them for having passed over many of the obvious faults of the preliminary manuscript which they had for reading and for having constructively discussed those points which were intended as contributions. I feel strongly, as does Professor Cole, that much that is without doubt essential can be learned about hearing by a further and more discriminating study of the more easily handled tissues of lower animals. Professor Davis points out the uncertainty of quantitative results obtainable from Chauchard's data. This question is also raised from still another angle in the manuscript, and I should like to point out that the object has been to introduce to ear specialists the possibility of dividing the time of reaction of a tissue into a physical and a metabolic phase, and to describe an approximate method of calculating the first. This concept may be applied to any measurement. Dr. Lewis' question concerning the mechanics of hearing leads directly to a rather copious philosophy which cannot be adequately treated in a short discussion. This question has been considered in a number of past meetings and printed in the transactions.

Tuesday Afternoon Session, May 17, 1932

The Relationship of Flora of the Upper Respiratory and Alimentary Tracts to Infections of the Mastoid, with Particular Reference to the Epidemiology of Mastoiditis. Dr. Samuel J. Kopetzky, New York.

In a study embracing a period of six years, the total admissions to Beth Israel Hospital were analyzed to determine the incidence of otitic disease, the ratio of acute to chronic cases, the percentage of mortality and the reasons for the seasonal and yearly variations in the statistics obtained. Subcultures were made, according to the Holman differential sugar technic, of the infecting micro-organisms in all otologic cases, and the occurrence of the various streptococcus types found in otitic lesions was compared with the incidence of the same types in all other pyogenic infections in patients admitted to the hospital during the period of observation. Data of considerable prognostic value were obtained.

An appreciable increase in the incidence of otitic infections was found every three years. These infections start in November and December, increase gradually in January and February and reach their maximum in March and April, declining sharply thereafter until their minimum is reached in August and September. The chronic infections follow the acute ones, reaching their maximum in the summer and early fall, when the general incidence is at its lowest.

The mortality rate is highest in the summer, when the general incidence is at its lowest. The ratio of acute to chronic cases does not explain this; rather it appears to be the usual reversal of the relationship of morbidity to mortality which is seen at the end of every epidemic.

It was found that the yearly mortality rate was not constant, although the average for the entire period was 5 per cent. While Streptococcus haemolyticus

was found to be the infecting agent in about 90 per cent of all cases, its presence did not explain the variations in mortality. Using Holman's technic and classifying the organisms present on the basis of their sugar-fermenting, as well as hemolytic, properties, we found Streptococcus haemolyticus-pyogenes to be the cause of almost 80 per cent of the mortality from infections of the mastoid. Streptococcus haemolyticus-infrequens was the type most frequently encountered in chronic mastoiditis.

Study of the yearly variations of the streptococcus types in mastoiditis revealed an orderly sequence in their periodicity suggestive of recurring cyclic changes. We believe that a major cycle embraces from five to six years. The existence of such a cyclic change in streptococcic types is borne out by the findings for all other metastatic foci as well as otitic infections. The parallelism between the graphic curves for mastoiditis, for other diseased centers and for the exposed mucosa of the upper respiratory and alimentary tracts prompts the inference that the last named may be the major source of all metastatic infections.

The fact that certain viridans types of streptococcus were regularly the precursors of hemolytic types with the same sugar-fermenting properties suggests the possibility that viridans forms may change into hemolytic types. The time required to bring about such an alteration appears to be a year.

DISCUSSION

DR. J. G. DWYER, New York: Our results tally, in the main, with Dr. Kopetzky's work, although we have not done such careful subculturing. Of course, we all recognize that the hemolytic organisms play the major rôle in infections of the middle ear and the mastoid and in their ensuing complications, and also that there are certain years and certain months in the year when these infections seem to be greater in number and in severity. We have been accustomed to attribute these facts to an increased virulence of the organisms in different epidemics, which in the main seems to be true, although Dr. Kopetzky's classification tells us that it is due to the different types of streptococcus. I can see that subculturing will give definite information for indications for operation, as well as for prognosis.

DR. EDMUND P. FOWLER, New York: Not being a bacteriologist, I should like to inquire how it is possible to obtain cultures from the chronic discharges that do not show many forms of organisms? From the mass of material, how do you pick out a certain type and say that this organism is accountable for the chronic discharge?

DR. SAMUEL J. KOPETZKY, New York: I will answer Dr. Fowler first. The bacteria were taken from the middle ear at operation. When this was opened, we took a swab from a sterilized tube and wiped off the secretions. In the ensuing growth, saprophytes and contaminations were cultured; only streptococci were transplanted for culture.

In regard to the classification, it would be desirable if studies similar to ours were instituted in different sections of the country. We should then have a more complete survey of the incidence and mortality rates by competent observers all over the country.

CIRCUMSCRIBED ARACHNOID CYST GIVING SYMPTOMS OF AN ACOUSTIC NEUROMA. DR. WILLIAM V. MULLIN, Cleveland.

Two cases, which at operation were proved to be cystic tumors of the arachnoid, were studied at the Cleveland Clinic. A review of these two cases and of others in the literature has raised the question in my mind as to whether it might be possible to make, before operation, a diagnosis of cyst of the arachnoid from the history and a complete and thorough examination of the ears. There are not many correlated data in the literature.

A complete history, making a point of ascertaining the sequence of symptoms, together with a complete vestibular and cochlear examination, might do much

to make possible a better diagnosis. There may be a variance in the age groups of patients with acoustic tumor, and those with arachnoid cysts. Infection and injury may both be etiologic factors. The duration of symptoms may also be important.

In the first case studied, there was some hearing in the left ear (the diseased side), with prolonged bone conduction on that side, no responses from both horizontal canals and no ocular changes. If I were to encounter again a patient with such bizarre symptoms, I should be suspicious of disease of the arachnoid.

In the second case, there had been a sudden onset of symptoms, namely, vertigo, vomiting and tinnitus with complete remissions. Spinal puncture completely relieved all the symptoms at once. The eyegrounds and visual fields were normal; there was no spontaneous nystagmus, and some responses to caloric stimulation remained on the right side, with normal responses on the left. The latter observation ruled out acoustic neuroma, and while it seemed at the time that degeneration of the right eighth nerve might account for the symptoms, the history of the onset of symptoms while stooping, the severe vertigo followed later by tinnitus and the fact that two years after the onset of symptoms a spinal puncture gave entire relief from all symptoms for five months might have rather definitely ruled out such a diagnosis and might have aroused suspicion of the presence of an arachnoid cyst.

From the present meager data, I believe that it is impossible to form any conclusions, but it might be that further study would elicit information which might make possible a differential diagnosis in these cases. The subject has been given practically no consideration by otologists, and it would seem that there has been a decided neglect on the part of neurologists and neurologic surgeons to have complete functional examinations of the ear made before operation.

DISCUSSION

Dr. Lewis Fisher, Philadelphia: An experience with a large number of cases of lesions of the brain demonstrates more than ever the difficulties of intracranial diagnosis. Among the many pitfalls encountered, that of arachnoid cyst is by no means the least important, and Dr. Mullin, in calling attention to this lesion, is offering a timely contribution. While arachnoid cysts are apt to lead the examiner astray in many and various types of intracranial lesions, the author's choice of cases with the lesions really otologic in nature adds greatly to the value of his discussion. One cannot emphasize too frequently that intracranial lesions involving the cerebellopontile angles belong particularly to the field of otology, and that the responsibility of a correct diagnosis rests directly on our shoulders and not on those of the neurologist or the neurosurgeon. You will recall that in one of the cases presented by Dr. Mullin (not his own case), a submucous resection had been performed in the attempt to relieve the patient of his deafness and tinnitus, before the true nature of the lesion was discovered. With the essayist, I think that it is unfortunate that a complete examination of the function of the labyrinth, both cochlear and vestibular, is not performed as frequently as it should be.

Dr. Marvin Jones, New York: A caution should be uttered against advising lumbar puncture in cases of lesions of the posterior fossa, because this would mean exerting force on the medulla and might cause respiratory paralysis and death. A preferable procedure would be to trephine the skull and aspirate the lateral ventricle.

Dr. Stacy R. Guild, Baltimore: Cystic formations are frequently found in association with acoustic tumors. In two cases in my practice the tumors were so located in the internal meatus that they could not possibly have been seen during operation. In view of these facts, I should like to ask Dr. Mullin whether in the cases which he has called arachnoidal cysts simulating tumors of the cerebellopontile angle, he can be certain that there was not actually a small tumor in the internal meatus with secondary cystic formation.

DR. J. GORDON WILSON, Chicago: The differential diagnosis between a neoplasm at the cerebellopontile angle and a cystic formation in that area, in my experience, in extremely difficult. The symptoms presented are more or less common to both. This is not to be wondered at, since they are both largely traceable to pressure.

The symptoms of a cystic development are best understood from a study of the anatomic relation of Luschka's foramen and the prolongation through it of the choroid plexus from the fourth ventricle to the surface of the cerebellum and the nerves emerging from the pons and medulla. (Slides were shown which illustrated this relationship, from Poirier, Paul, and Charpy, A.: Traité d'anatomie humaine, Paris, Masson & Cie, 1901, vol. 31, pp. 133 and 152.) With these anatomic relations, it is easily understood how a localized subarachnoid cyst may be formed and how it can involve not only the seventh and eighth nerves but the sixth, ninth and eleventh nerves as well.

DR. W. V. MULLIN, Cleveland: Dr. Fisher, in the past, gave us something so definite on which to base a diagnosis of tumor of the cerebellopontile angle that when in any case the findings do not correspond to the symptomatology that he presented, it is sometimes hard to convince the neurologic surgeon that there is disease in this region other than an acoustic tumor, and that an exploration should be made. I am observing a case now, but several surgeons hesitate to operate because the typical angle syndrome is not present, and because the deafness is of the conduction rather than of the perception type. This particular phase is merely a coincidence. If they do not operate soon, it will be too late, for the man will be killed while driving his automobile, which he persists in doing despite severe attacks of vertigo.

I believe that patients with cysts of the brain have a protean symptomatology, with considerable variation in hearing and in labyrinthine response.

VIDIAN NEURALGIA: WITH SPECIAL REFERENCE TO THE EYE AND ORBITAL PAIN IN SUPPURATION OF THE PETROUS APEX. DR. HARRIS H. VAIL, Cincinnati.

This article will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. D. E. WISHART, Toronto, Canada: I am interested in disease of the petrous apex. I have two patients suffering from multiple osteomyelitis. In one patient hearing is lost in both ears and the vestibular responses have disappeared. The second patient has lost the entire function of the eighth nerve on one side, and on the other side he has a partial loss of hearing with a partial loss of vestibular response. The suggestion is that the focus of infection lies in the petrous apex. In the absence of signs in the middle ear, how can this condition be diagnosed? Has Dr. Vail in his study of disease of the petrous apex come across any cases similar to mine?

DR. HARRIS H. VAIL, Cincinnati: It is obvious that we must diagnose suppuration of the petrous apex early, and the significant pain back of the eye in these cases is a symptom which appears early. After all, it does not matter a great deal just how the pain is produced, so perhaps my paper may be splitting hairs. The important thing is the significance of the pain back of and around the eye on the same side as the affected ear.

I am unable to answer Dr. Wishart's question. The labyrinthine disturbance may be due to either a metastasis from the infection or a toxemia.

Tuesday Evening Session, May 17, 1932

TREATMENT OF OTITIC LEPTOMENINGITIS: A PLEA FOR INVESTIGATION IN TWO DIRECTIONS: (1) KUBIE'S THEORY OF FORCED DRAINAGE AND (2) DIRECT SURGICAL DRAINAGE. DR. PHILIP D. KERRISON, New York.

This outline of new methods in the treatment of otitic meningitis was presented because the surgical treatment of otitic leptomeningitis has produced only meager results. There is still a lack of a rational, clearly defined and generally accepted plan of surgical treatment which, in any large series of cases, can be depended on to save even a small percentage of lives. E. W. Day, in 1913, after an exhaustive study of this disease, concluded that "theoretically, surgery should give relief, practically, the mortality has not been changed by it."

In passing on to the most recent experimental work by Weed and Kubie, the author paused to criticise the work of Eagleton on a subarachnoid irrigation, on the withdrawal of spinal or cerebrospinal fluid, with replacement with Ringer's solution, and also on ligation of the common carotid artery, as procedures lacking a physiologic basis. He prefers to advocate therapy in the direction of forced drainage, or of direct surgical drainage, according to the studies of Kubie and Weed.

A brief description of these methods was given. Kubie's theory of forced drainage is as follows: The cerebrospinal fluid is described as a dialysate or a filtrate from the capillaries of the choroid plexus and, in small amounts, from the perivascular and perineural spaces of the central nervous system. Due importance is given to the latter source of the filtrate.

Further, the cerebrospinal fluid is stated to be in osmotic equilibrium with the blood. The injection of distilled water or a hypotonic solution or the drinking of large amounts of water causes increased intracranial pressure and an increase in the production of spinal fluid. If, however, the injection of hypertonic solution permits loading of the blood with salts, osmosis works the other way and results in a diminished brain bulk and a diminished production of cerebrospinal fluid. Thus, intracranial pressure and the production of cerebrospinal fluid may be changed at will in either direction by dilution or concentration of the blood salts.

Weed, in 1923, experimented on dogs, in order to determine the source of the cerebrospinal fluid. He found it to be in the choroid plexuses and the perineural and perivascular spaces of the brain.

Kubie's work was based on the therapeutic possibilities of Weed's experiments. He worked first on dogs, and instituted continuous drainage from the cisterna magna, making pressure observations through a trephine hole in the parietal bone. The subarachnoid space was not entered. The cisterna magna was connected with a second manometer, and by this means pressure could be recorded either when there was a flow from the cisterna magna or when it was shut off. Changes in pressure varying from 2 to 5 mm. were observed with each heart beat; greater changes were caused by inspiration and expiration; the pressure was reduced during the outflow from the cisterna, and changes in the pressure resulted from injections of hypotonic solutions.

Kubie states that in inflammatory conditions of the central nervous system and of the meninges, the perineural and perivascular spaces are lined with newly formed cells, many of which are lymphocytes. Following intravenous injections of hypotonic solutions accompanied by continuous drainage of the cerebrospinal fluid, masses of these cells may be seen post mortem to have been extruded into the subarachnoid space.

Dogs on which these experiments have been repeatedly made at intervals of only a few weeks have made apparently permanent and perfect recoveries.

The basis of the theory, then, is that the perineural and perivascular spaces, which during meningeal inflammation are the supply posts for the enormous increase in the number of lymphocytes, are also the storage points of pathogenic micro-organisms, and that since these cell-filled and infected crevices are in part

the normal starting points of fluid production or dialysis, a stimulated increase in fluid production may rid the central nervous system of toxic matter through an autogenous cleansing of its perineural storage points. It is urged that a group of men of scientific attainments and technical skill, working in a well equipped laboratory, should further elaborate this work in its therapeutic aspects.

DISCUSSION

DR. ARTHUR B. DUEL, New York: Dr. Kerrison's paper is a strong plea for more research on the problem of meningitis. He advocates the method of Louis Weed, who has discovered a simple method of reducing intracranial pressure. Kubie's careful experiments on animals, pointing to the value of intrameningeal drainage, may be shown to have great clinical importance. Meningitis is not a peculiar disease, belonging only to the meninges, but is one that is applicable to the same broad surgical principles observed in other parts of the body. An additional difficulty, however, is that the meninges are inclosed in a bony capsule. Methods of craniectomy at the present time are not greatly in advance of neolithic surgery. It is urgent, therefore, that craniectomy should be made the subject of careful experimentation and improvement in technic.

We must bear in mind that meningeal inflammation may be local or diffuse; it may be intradural without involving the arachnoid spaces, or may involve both. These varying types present different degrees of menace to the patient. The simpler forms, if the patients are operated on in time, are within our power to check, but may, if the patients are not operated on, progress into the graver forms beyond our power to check.

We must emphasize the following points: (1) early diagnosis, (2) immediate operation, (3) adequate drainage and (4) renewed effort for an improved technic.

DR. LAWRENCE S. KUBIE, Boston: In regard to the question of so-called "forced drainage," the following points of meningeal drainage should be borne in mind:

1. Early drainage is necessary, before walling off occurs in the intracranial spaces.

2. There is a necessity not only of surface drainage, but of drainage of the depths of the central nervous system as well.

3. Opening of the skull may cause collapse of the meninges or a mass settling of the whole brain on the base, causing obstruction to the outflow of fluid.

4. This collapse can be prevented by forcing the production of cerebrospinal fluid.

5. Normally, cerebral pressure is maintained by a balance of the fluid transuded through the capillaries and that reabsorbed by the venous capillaries. If, however, intracranial pressure is reduced to atmospheric pressure by drainage and at the same time the production of cerebrospinal fluid is stimulated by a lowering of the osmotic pressure of the blood, we can obtain fluid movements from the depths of the brain to the surface and out through the operative drainage tract.

6. There are various methods of drainage, none so far perfect, but the most important seems to be lumbar drainage with the head high, so as to drain the whole tract.

7. Drainage alone cannot effect a cure, but must be supplemented by adequate surgical procedures and the promotion of fluid.

8. The promotion of fluid is best effected by intravenous injections of a hypotonic solution. The injection is made slowly to prevent overloading of the circulation with a strain on the heart.

9. Difficulties in the way of drainage are: (*a*) the thick gelatinous exudate which forms in the meninges and (*b*) the formation of pockets of infection. The first difficulty will have to be solved by proper dilution of the exudate. The second point will depend on individual procedures. Recently, Dr. Leon Cornwall

of New York used the injection of air to break up obstinate adhesions, with the result of successful evacuation of a large quantity of pocketed pus.

10. The nasal cavity can be considered as a means of possible approach to the base of the brain. This is a pathway by which a continuous system of forced drainage might be established under the proper technic.

DR. PHILIP D. KERRISON, New York: Dr. Kubie's discussion was particularly interesting for the reason that what he says with regard to forced drainage may be regarded as authoritative. Dr. Duel referred to the surgical analogy between the treatment of otitic meningitis and that of suppurative processes in other parts of the body. In theory, at least, that is a point of considerable importance, for if we run over mentally all the known lesions due to infection, we shall find it difficult or impossible to recall a single suppurative lesion, unless it is that of suppurative endocarditis, which is not in some degree amenable to surgical treatment. Is suppurative leptomeningitis the one other exception, the one other suppurative lesion which will forever remain beyond the scope of surgical therapy? Combining logical surgical methods with the forced drainage theory of Kubie, I believe that the reverse will prove to be true.

Dr. Eagleton speaks of different types of meningitis, some of which he says, dogmatically, are hopeless. I should call Dr. Eagleton's attention to the fact that at the present time experiments and investigations in this field are being carried on by several highly scientific groups, and that it is highly unscientific to state in advance what the ultimate results of these experiments will be.

With regard to ligation of the carotid, Dr. Eagleton brings to the support of the procedure simply his unsupported work and belief that it is a wise therapeutic measure. He thinks that he has beneficial results from it in cases of otitic meningitis. Some of us hold exactly the opposite opinion.

Naturally, the first and most obvious argument against it might be the fact that it violates the generally accepted physiologic law that an unimpeded blood supply to a diseased part is one of nature's chief instrumentalities for bringing about resolution and recovery. I prefer, however, to base my argument against it on somewhat different grounds.

Both Dr. Eagleton and I are interested in the treatment of meningitis, but neither he nor I would claim to be an authority on the subject. No man is. Neither he nor I, therefore, can expect a purely personal opinion or even a personal statement of experience to carry much weight or to survive if it is in opposition to the consensus held by scientific workers and physiologists generally. Modern scientific thought is overwhelmingly behind the thesis or proposition that the cerebrospinal movement is in itself the instrumentality through which, if ever, purulent meningitis will be brought under therapeutic control.

In one of Dr. Eagleton's papers he discussed at some length the so-called "cerebrospinal circulation" and the agencies through which it is initiated and maintained. His conclusion, which of course is the obvious one, was that next to the hydraulic pressure of continuous production, the movements of the cerebral fluid are maintained chiefly through the bulk movements of the brain itself, which movements of the brain are activated chiefly and probably solely by arterial pulsations. When, therefore, he proposes as a therapeutic measure in purulent meningitis to ligate the common carotid, he suggests a procedure which should tend: (1) to diminish the production of fluid in the ventricles and in the perineural spaces of the central nervous system; (2) by reducing arterial pulsations, to inhibit or reduce the movements of the brain by which the cerebral fluid is kept moving, and (3) as a by-product of the mechanical result of 1 and 2, to promote cerebrospinal stasis in the germ-laden cisterns at the base of the brain and in all the perineural spaces throughout the central nervous system. Viewed in this light, ligation of the carotid seems to be not only sadly illogical, but a violation of modern scientific thought and theory, which is, to say the least, somewhat archaic.

Dr. Eagleton states that a prerequisite to successful treatment is the determination of the pathologic changes by which the infection has taken place. It would,

of course, be highly desirable if this could always be done. But each of us has, in the course of our otologic experience, seen and studied many cases of otitic meningitis, and I think that the members of this Society will bear me out in the statement that the cases in which the pathway of infection can be definitely determined are the exceptions rather than the rule. Dr. Eagleton is, therefore, insisting on what we all know to be, in the vast majority of cases, an impossibility.

Wednesday Morning Session, May 18, 1932

EXPERIMENTAL INVESTIGATION OF THE PHYSIOLOGY OF THE EAR, USING THE METHOD OF WEVER AND BRAY. DR. S. J. CROWE and DR. WALTER HUGHSON, Baltimore, Otological Research Laboratory, Johns Hopkins University.

At the meeting of the American Otological Society in June, 1931, the authors presented the results of their first year's work on the normal and pathologic physiology of the ear as studied by the method of Wever and Bray. During the first year only the simplest equipment was used, and the conclusions drawn were largely subjective impressions. At this meeting the equipment used for the more accurate recording of experimental results was described, and charts were shown of the relative intensity of the transmission of speech and of pure tones (from 250 to 4,000 cycles) before and after the production of experimental lesions of the various structures in the middle ear. Both air and bone conduction have been studied with this method.

The apparatus is described in detail by E. G. Witting in an article now in press. A general description of the first year's work was published in the *Zeitschrift für Hals-, Nasen- und Ohrenheilkunde* (**30**:65, 1931) and in the *Proceedings of the Inter-State Post-Graduate Medical Assembly of North America* (1931, vol. 7). The plan of investigation of the Otological Research Laboratory and some of the more important physiologic conclusions from the study of the human material and the animal experiments were presented before the Harvey Society in New York in January, 1932. The experiments on the effects of immobilization of the membrane of the round window were presented by Walter Hughson before the American Association of Anatomists in March, 1932, and will be published in the *Annals of Otology, Rhinology and Laryngology.*

DISCUSSION

DR. E. G. WEVER, Princeton, N. J.: I wish to speak chiefly on a point of method, in regard to determinations of intensity, which, though closely comparable to that of Dr. Crowe, has a difference in the switching arrangement, which we consider advantageous and by which one observes alternately the responses from the nerve and the standard sound, the arrangement being such that when the standard sound is equal in loudness to the sound from the animal, one hears a continuous sound instead of a varying one as the switching is made. The two sounds are observed against the same background of noise constantly present, and quantitative determinations may be made with considerable precision.

MR. R. L. WEGEL, New York: The questions raised by Dr. Crowe's work are principally questions of mechanics. He is studying the performance of the middle ear, using the nerve tissue more or less as he might use a microphone to detect vibrations. Having thus largely limited himself to a study of the middle ear, his results bear on more specific questions, and so far as he has actually eliminated the nerve tissue, his results can be discussed in terms of well known principles of a quantitatively unknown problem.

Most of the observations which he has reported are in accord with what is to be expected on the basis of a crude knowledge of the mechanism, but there

are certain results which seem to contradict this simple type of reasoning. This is by no means surprising, as any one who has had experience in the quantitative design of acoustic devices will know. From a rigorous standpoint the mechanism of the middle ear may be thought of as constituting six possible channels for transmission of energy from the ear to the labyrinth, each channel corresponding to some combination of the six possible motions of each one of the elements of the chain. The ossicles are so attached that these six ways of transmitting energy are not independent. This mechanism is roughly analogous with six telephone lines, all connected to a transmitter at one end and a receiver at the other end. These transmission channels, in order to carry out the analogy, must be close enough together so that there is a considerable amount of "cross-talk" between them. When Dr. Crowe applies a pledget of cotton to the round window he must, rigorously speaking, affect all six of these channels, and thus, while it seems that he is providing one mechanical constraint, he is actually applying a minimum of six constraints. If the quantitative constants of the middle ear were accurately known, they would no doubt show that of the six channels only one or perhaps two are of any practical importance in transmission of sound except possibly in certain types of pathologic conditions. Each of these channels is of the general type of "band pass" filter which transmits sounds within a certain frequency range more efficiently than others, and the combination of the six channels is also a "band pass" structure. This follows from a mere inspection of the parts and is in accord with the observed fact that the ear is most sensitive within a frequency range of from 1,000 to perhaps 5,000 cycles. Whether or not a working concept of the mechanism is most advantageously stated in such terms as I have given, the fact remains that in order to find out what to expect when a given lesion exists, it will be necessary to solve the problem of mechanics as I have stated it. When this is done, there will be no unexpected results obtained by mechanical blocking of certain parts. Dr. Crowe frequently speaks of the action of the pledget of cotton on the round window as blocking its motion. This is an unfortunate term to use, for it suggests that the round window is thereby prevented absolutely from moving. It will be fairly obvious that the addition of a pledget of cotton cannot have an effect greatly different in order of magnitude than that of the cochlea itself. Dr. Crowe's results, of course, have, as I understand him to say, a direct outlet in the treatment of hearing defects quite regardless of any theory, but those of us who are primarily interested in finding the mechanism will be much interested in following Dr. Crowe's continued work along this line.

Dr. D. Harold Walker, Boston: For years, through the suggestion of Dr. Clarence Blake, I have been placing cotton pledgets in the niche of the round window for the purpose of improving hearing. The hearing apparently is improved by the blocking of the round window and by not allowing any vibrations which go to the organ of Corti by means of the stapes to escape.

Dr. Eugene R. Lewis, Los Angeles: I do not see how hearing was improved by blocking the round window. Did I understand that correctly?

Dr. S. J. Crowe, Baltimore: Yes, the hearing was improved.

Dr. Edmund P. Fowler, New York: Did Dr. Crowe notice any difference in the quality of the sounds after blocking the round window? If this was due to reflection of sound, there would be some slight alteration of the tone quality.

Dr. S. J. Crowe: The sound was much clearer and more distinct. The increase was from 3 to 15 feet.

There is a simple experiment that demonstrates the improvement in bone conduction. In the cat the bulla and middle ear are opened through an incision in the neck; air conduction and bone conduction are tested and charted as described in previous publications. The middle ear and bulla are then filled with mercury. This fixes both the stapes and the membrane of the round window. Under these conditions air conduction is nil, but bone conduction is actually and measurably improved.

ELECTRICAL PHENOMENA IN THE AUDITORY MECHANISM. DR. L. J. SAUL, DR. H. DAVIS and DR. M. H. LURIE, Departments of Psychiatry, Physiology and Otology, Harvard Medical School, Boston (by invitation).

A method employing modern neurophysiologic technic is used for the study of electric acoustic phenomena. Two effects are distinguished: true nerve action currents and a spreading electrical effect from the cochlea.

The true action currents have been traced along the auditory tracts from the eighth nerve to the auditory radiations. They are sharply localized, follow contralateral tracts, are reversibly deleted by anesthetics and disappear immediately on sectioning the eighth nerve or at death. On using musical tones as stimuli, the pitch applied to the ear is recovered only up to about 1,000 double vibrations per second. Words are not intelligible, probably because the higher pitches are lost.

The "spread," in contrast to the action currents, is a diffuse electrical broadcast which probably originates in the cochlea and which spreads through all the tissues of the head. It is probably in no way concerned with physiologic transmission from ear to brain. It shows a point of maximal response in the posterior groove of the subarcuate fossa. It is much more resistant to anesthetics than are action currents. It may persist for several minutes after death. In one experiment it gradually faded out over a period of twenty minutes after complete cutting of the eighth nerve and the cochlear artery. In response to musical notes applied to the ear, tones may be recovered up to at least 4,000 vibrations per second. The lower limit, at a given intensity, is not so low as for action currents. Words are reproduced clearly.

Some preliminary technical observations were presented. The action current appears in the eighth nerve, from about five to eight ten-thousandths of a second after the peak of the sound wave. Stimulation seems to occur at the peak of the sound wave. The intensity threshold for action currents appears to be lower than that for spread. Their relation to intensity of stimulating tones seems to be logarithmic, while that of spread is linear. Strong harmonics are evident in the spread wave at certain frequencies.

A cat exposed to a tone of 600 double vibrations for three months and another for six months showed no tone islands. The latter cat showed a definite maximum at 600 double vibrations in the action currents from the brachium and auditory radiations, but no maximum from the eighth nerve or spread.

The authors conclude tentatively that the upper limit of frequency of action currents in a nerve fiber is probably not over 1,000 per second.

DISCUSSION

DR. LORENTE DE NO, St. Louis: Dr. Saul, Dr. Davis and Dr. Lurie have determined the frequency of the potential changes in different parts of the acoustic tract. I should like to know whether the frequency of their "true action currents" is the same in the eighth nerve as in the medulla and in the midbrain, or whether there is an indication of change of frequency when the impulses pass through one of the nuclei of the acoustic tract (the ganglion ventrale and tuberculum acusticum, the oliva superior complex, the posterior colliculus and the corpus feniculatus internus). If the electrical changes recorded by the authors are true action currents, it is most likely that they change their frequency when the impulses pass through the different nuclei. In the cochlea there are several different kinds of endings, of which the most important are the fibers for the internal hair cells, of which it may be said that there exists a fiber for each hair cell or for a few closely lying cells, and the spiral fibers that are connected with numerous external hair cells. Any vibration of the membrana basilaris will set up impulses in many fibers, and, of course, not simultaneously. A few fibers that each conduct a small number of impulses per second can give the picture of impulses traveling at a high frequency if the fibers are not synchronized, as most likely happens in the cochlear nerve.

DR. D. HAROLD WALKER, Boston: If the physiologist can tell us how the sound is taken from the organ of Corti to the cerebrum and if the physicist can devise a method by which sound can be transmitted to the organ of Corti by some other method than through the middle ear, then the problem of deafness due to adhesive changes in the middle ear might be solved.

DR. JOHN TAIT, Montreal, Canada: Dr. Saul said that the electric charge reached the brain and spread all over, but the action currents proper were quite localized. He traced them to their final termination in the cortex. Is the rhythm retained in spite of the interposition of other nuclei on the pathway? Dr. Saul said that he could put a needle in subcortically and could pick up action currents at their termination. His exposition concerned particularly the preliminary nerve conduction stage, rather than the final cortical stage.

DR. E. G. WEVER, Princeton, N. J.: In regard to the stimulation-deafness problem mentioned by Dr. Saul, I can add two more negative cases. We stimulated cats with very loud sounds for three weeks, but on testing them found no evidence of modification of sensitivity. We suspect, on the basis of previous studies in this field, that certain animals are more susceptible to the destructive action of sounds than others. Horton, working in our laboratory, is at present developing the conditioned response method with guinea-pigs to a point where audiometric curves can be taken. In the seven animals thus far tested, he found the hearing much like that of man, with no greater differences than would be apparent in seven human subjects selected at random.

DR. M. A. GOLDSTEIN, St. Louis: If the physiologist places the maximal rate of vibrations at 1,000 when he measures nerve action current, how can we hear 30,000 double vibrations? If he measures 1,000 double vibrations or less through any nerve impulse, what right has he to presume that any other impulse may be measured in any other way?

DR. L. J. SAUL, Boston: Our observations support Dr. Crowe's, and we believe that he is working primarily with spread, which, in the vast majority of cases, is an excellent index of what the animal is hearing. In answer to Dr. de No, we get the same frequencies up to the acoustic radiations. What happens in the cochlea when sound is transformed into electrical impulses is only hypothetic, and more than one mechanism may be found to fit the facts. Dr. Goldstein's question plunges us into the problem of auditory theory. We can outline possibilities, but cannot say what actually happens.

RESEARCHES ON LABYRINTHINE REFLEXES. DR. RAFAEL DE NO, St. Louis.

Dr. de No gave a summary of the results of his study of the anatomy and physiology of the labyrinth and vestibular apparatus. In the first part, he described the most important characteristics of the vestibular reflexes, recorded by the ocular muscles of the rabbit (postural reflexes, rotation reflexes and reflexes due to excitation of the individual semicircular canals).

In the second part is described the anatomy of the peripheral and central endings of the vestibular nerve.

In the third part, he mentioned some experiments on the physiology of the vestibular pathways in the medulla and midbrain and presented two diagrams which may explain two of the most important features of the vestibular reflexes: the after-discharge and the appearance of the quick phase of the nystagmus.

DISCUSSION

DR. JOHN TAIT, Montreal, Canada: Dr. de No has been working on the equilibrial part of the labyrinth along different lines, but on lines that give results similar to those obtained by Dr. McNally and myself. Dr. McNally has cut nerves without interfering with the membranous labyrinth, so that we can have a frog in which one, two, three or, in fact, any number of canals are lacking. He can leave a single canal or any combination of canals intact. Thus, a nega-

tive type of experiment in the one case can be checked by its corresponding positive in the other. We have found that the whole apparatus of the labyrinth, the six canals and the two auricular maculae, works as one unit in maintaining and controlling equilibrium.

Dr. J. Gordon Wilson, Chicago: Dr. de No's paper is a physiologic investigation of the labyrinthine rôle in compensation. During movements of the head in space, the eyes of normal persons make adequate compensatory movements necessary to ensure precise vision. In the action of the ocular muscles necessary to give this equilibrium in spite of bodily movements, the labyrinth plays a part.

The fixation of the eye for vision is a perfect mechanism, and in all voluntary movements the six ocular muscles in each eye take a part as agonist, antagonist and fixation muscles. Also, in movements of the eyes from labyrinthine stimulation, all the muscles participate to give the appropriate deviation.

When we turn attention to the labyrinth and inquire what is happening, we find ourselves debarred from direct observation. Maxwell, in the dogfish, could by direct observations on the essential parts of the labyrinth see what he was doing. In the frog and birds, Tait and McNally are coming close to this. However, in mammals we are stimulating a peripheral organ not so directly available, and our physiologic conception of what occurs is influenced more or less by a deduction from studies on lower vertebrates based on mammalian anatomic knowledge.

While it is agreed that there are two muscle reactions from irritation of the labyrinth—(*a*) kinetic or dynamic and (*b*) static or postural—the anatomic position of these reactions, as well as the physiologic connections involved, is disputed. Maxwell showed in the dogfish the ampullae alone suffice for all the dynamic and all the static postural functions of equilibrium originating in the ear, and that the otolith organs of the utricle suffice for all static and dynamic functions of equilibrium except the response in a horizontal plane. Magnus believed that the dynamic reaction originated in the ampulla of the canal, and that the excitation of the maculae of the utricle and saccule gives a tonus or static effect. Tait and NcNally believed that the semicircular canals are not involved in static reactions; they respond only to acceleration and give only compensatory movements; the utricle is the apparatus for static equilibrium. Dr. de No's experiments tend to support those of Maxwell.

To me the most interesting part of the paper has been the neurologic side. The diagrams of the nerve endings are interesting and convincing. Dr. de No's diagram to explain why the nystagmus outlasts the excitation of the labyrinth (which it may be remarked is but one example of a phenomenon so characteristic of reflex contractions) appears to me as satisfactory as any yet offered and well in keeping with the results of Ranson's experiments.

Professor F. Parker, Boston: Trophic transmission over nerve fibers probably takes place by the passage of substances along the nerve. Such substances may escape at the nerve terminals, and in this way the nerves may become secretory. Probably this function permeates the whole nervous system, the secretory activity taking place at the synapses. Chromatophores respond to such secretory material long after the true action of the nerves has ceased. The retardation mentioned by Dr. de No may thus possibly be approached.

Dr. Rafael de No, St. Louis: If that were true, it would settle the discussion. There would be some mechanism to take away or compensate for the secretion. We need both substances to complete the explanation.

We must, of course, be careful in translating the results of experiments on rabbits to the physiology of human beings. The eyes of the rabbit show strong tonic reflexes that in man are almost missing, and on the other hand, the rabbit does not have optic reflexes of any importance. Then we must consider that the labyrinth is prepared to produce normal reactions only when it is excited under normal conditions. The nystagmus compensates the rotation of the head only when the rotation is a physiologic one, i.e., when the rotation corresponds

to any of those that are used by the rabbit itself. After-nystagmus is never physiologic. In my papers I have described a "physiologic rotation" that sets up rotation nystagmus, but not after-nystagmus.

The diagrams attempting to explain the production of after-discharge and those of the quick phase of the nystagmus have been developed in many directions; they are perfectly consistent with each other if we consider that the neuron chains may have quite different thresholds, and, therefore, that they may need different times for setting up a discharge. The experiments of Dr. Tait and Dr. McNally will certainly be of great importance for the physiology of the labyrinth. I am glad to have heard their results in advance.

I agree with Dr. Parker that chemical changes must be the primary cause of most of the nerve phenomena. The theory of an exciting and inhibitory substance producing antagonistic effects would explain most of the results of the experiments that I have carried out.

Wednesday Afternoon Session, May 18, 1932

THE USE OF VARIOUS TYPES OF AUDIOMETERS IN CLINICAL WORK. DR. C. C. BUNCH, St. Louis.

Clinically, hearing tests are designed to assist in diagnosis and to help determine the effects on hearing of any treatment which may be instituted. A clinic in which teaching is a primary function must also take into consideration the training of those being taught (interns, assistants and others). More than this, the correction of false ideas gained by early methods and inadequate facilities as well as the acquisition of new facts should continually be kept in mind. The otologic clinic in Washington University, for the purpose of development along these lines, has secured and is using for auditory acuity tests the various types of audiometers which are available. The standard voice and tuning forks tests are also used; comparisons are made, and variations are noted. This study is a report of this more or less general type of work.

DISCUSSION

DR. GORDON BERRY, Worcester, Mass.: The manufacture and sale of audiometers by large concerns do not always adapt to specific needs. A laboratory group is more able to adapt its instruments to required changes. The Bell Telephone Laboratories have done the medical profession an immense service in carrying out testing on a large scale. Different types of audiometers have their limitations; thus, the 3-A audiometer is a simple testing device, but would probably serve better if it offered more amplication with a mean low, high and middle tone, instead of the universal tone it furnishes. The 4-A audiometer is admirable for testing numbers of school children, and is useful in doctors' offices and in clinics. The 2-A audiometer is made for the otologist. It is quicker and simpler than the 1-A, but it is apt to get out of order and takes time and money to repair. If we could attain a wider usage, a cheaper production and a more efficient servicing, more men would use audiometers and realize their indispensible value to the medical profession.

DR. DOUGLAS MACFARLANE, Philadelphia: Audiometers are a valuable addition to our armamentarium, and I do not think that we can do without them. Dr. Bunch has shown that they are practical instruments for routine use, and not merely instruments for scientific investigation, but the instruments themselves do not supply a knowledge of how to use and to interpret them.

Faults in technic should not be blamed on the audiometer. When we are dealing with the deafened class, we find such great differences between single tone testings and speech hearing testings that it is impossible to explain them. One

cannot tell from a 2-A audiometer record what speech hearing may be. The hearing of single tones is a relatively simple mental process as compared with speech-hearing and interpretation; the latter is related to the central perceptive sphere. It is also highly important to note the type of mentality of the patient being tested. Is he alert and intelligent or slow, uncertain, cautious or illiterate?

Another practical point is the checking up on gradual changes in progressive deafness. While it may not be possible to improve hearing conditions, much can be done from the point of view of psychologic and economic readjustment. If deafness is progressing to a point where there will be incapacity for occupation, the change must be anticipated.

DR. EDMUND P. FOWLER, New York: The results of Dr. Bunch agree with those of us who have done audiometric work. The time has gone when it could be supposed that we could make a diagnosis on one set of tests alone. There is no one instrument with which we can make a complete diagnosis.

DR. C. C. BUNCH, St. Louis: In regard to the relation of these tests to hearing aids, it seems to me that in the type of deafness in which the patient hears low tones fairly well and high tones poorly, it is necessary to have some type of device which will amplify high tones more than low, or a filter in an amplifying circuit which will cut down the low tones which need less amplification. This can be done in the laboratory, but such devices should be available to the otologist. There is no doubt that the psychic element has much to do with the benefits received. With the proper attitude of mind, the patient will listen more attentively. Women will often refuse to wear a hearing device because they think that it makes their deafness more conspicuous.

THE PRESENCE OF PHAGOCYTIC CELLS (HISTIOCYTES) IN THE AURAL MUCOSA. DR. RALPH A. FENTON, with the collaboration of DR. OLOF LARSELL, Portland, Ore.

Aural histopathology in the past was concerned largely with epithelial and bony changes. The rôle of connective tissue in immunity was neglected. Difficulties of securing and staining material were present.

So-called "reticulo-endothelial" connective tissue cells, ordinarily quiescent, are fixed elements capable of phagocytosis when needed. They are demonstrable in tissues through the ingestion of vital stains. These cells, "histiocytes," were not mentioned in otologic histopathology until recently.

Submucosal connective tissue in childhood is thick, becoming granulation tissue when irritated and overgrown by ciliated epithelium from the mouth (Wittmaack). Meyer disagrees that a tubal connection is necessary, mentioning ciliated change at a distance. The thin connective tissue of the adult becomes fibrotic with irritation. The eustachian tube is cilated; there is much adenoid tissue near the pharynx.

Demetriades (1927) stained connective tissue intra vitam with pyrrole blue. Russi (1930) demonstrated the distribution of histiocytes in guinea-pigs, using trypan blue. Schwarz (1931) found that constitutional peculiarities influence the amount and distribution of connective tissue in fetal and infant temporal bones. Using Maximow's stains on 202 bones, he demonstrated 10 per cent of connective tissue cells to be histiocytes before the fifth fetal month, increasing rapidly in proportion after the appearance of foreign matter in the tympanic cavity at that time. Histiocytes are numerous at term.

Meyer (1931) considered "myxomatous" or "fetal" tissue to be only edematous connective tissue similar to that of the nasal sinuses and polypi. Our material on cats, vitally stained with trypan blue, showed few but constant histiocytes along the eustachian tube and still fewer near the tympanic capillaries. Normal adult human material shows fairly numerous histiocytes in the fibrous submucosal tissue of the tube and fewer in the tympanic submucosa. A vascular aural polypus due to long-standing chronic suppuration showed numerous histio-

cytes. Fibrous changes are more marked in granulations adjacent to cholesteatoma, but active histiocytes are also observed. The rôle of plasma cells is not yet determined.

Histiocytes first act as phagocytes, and then form fibrous tissue. The influence on the pathology of the ear is obvious, and suggests extensive experimental work as well as examination of carefully fixed human material prepared with stains which demonstrate these structures.

DISCUSSION

DR. L. W. DEAN, St. Louis: I should like to ask one question. How is it that Dr. Fenton is so certain that the stain he is using proves that the cells are histiocytes?

DR. J. GORDON WILSON, Chicago: The investigations now going on regarding the rôle of the histiocytes unveil a little more the secret of resistance of tissue to disease. Dr. Fenton has, on several occasions, added to our knowledge. We hope that he will continue this work. The wandering and sessile histiocytes widely distributed about the body are no doubt as active in diseases of our specialty as they are in general maladies. Regarding their rôle, we can ask a number of unanswerable questions, but I am content to wait until Dr. Fenton can present us with more reliable information.

DR. RALPH A. FENTON, Portland, Ore.: If the histiocyte gets too full of bacteria, the cell dies. It is taken up by other cells, broken down or carried away. We checked this work on the frontal sinus mucosa of cats. It is probably the same in the eustachian tube. Dormant cells become active and take up their histiocytic function. In one specimen which was carefully dissected, there were extrusion and diapedesis into the lumen of the cavity. In Hilging's moving pictures of mucosal cells, it is shown that débris is absorbed in various ways by the blood stream or by polymorphonuclears.

We should definitely agree on terms to be used in the application of newer staining methods. The motto for the cytologist of the future will be *semper mutable*.

DR. L. W. DEAN: How soon do you kill the animal after using the vital stain?

DR. RALPH A. FENTON: We have used different times, depending on the size of the animal and the character of the experiment, but generally within from twenty-four to fifty-eight hours.

IMPROVED HEARING IN FIXATION DEAFNESS: CASE REPORTS; COMMENTS. DR. EUGENE R. LEWIS, Los Angeles.

Several unusual observations new to me are noted in cases of progressive deafness, and suggest that recovery of a useful degree of hearing may be obtained. These cases of bilateral, progressive deafness of from fifteen to twenty years' duration which are on the increase have, by systemic measures only, shown striking improvement.

These facts suggest certain clinical and biologic considerations, namely: (1) Deafness was produced by changes in the tissue which altered the shape, size or texture of the bony or ligamentous structures at or near the oval window; (2) clinical evidence shows a gradual increase of such changes in the tissue; (3) the bilateral nature of this deafness suggests a predisposition to such changes; (4) the bilateral nature of this deafness points to activating systemic causes, such as puberty or pregnancy, and (5) attempts to alter the constitution of the body tissues in long-standing otitic conditions have given striking results, such as vertigo, nausea, cessation of tinnitus, increased hearing acuity accompanied by general systemic changes in body weight, blood pressure, blood counts and metabolic rates.

The explanation of these phenomena must be sought in biologic changes which occur after the tissue and nutritional factors have been altered. There must be, also, intrinsic and extrinsic factors which take part in the changes in the tissue under consideration.

The nature of some of these biologic factors may be described as follows: Experiments on regeneration of cell structure in plants and animals, by various workers, have shown that a water-removable substance takes part in normal cell division and that oxygen must be extracted from the air to facilitate cell division. Also, in growing organisms, action currents flow from older to younger growing portions of an organism. Action currents of abnormal cells (such as tumor cells) differ from those of normal healthy growing cells. In communities of living cells, differences of potential level are manifested as action currents. In polycellular organisms, electropolarity determines their direction of growth. If the polarity is reversed, the direction of growth is reversed. Heat and chemical action are also seen to have their influence on cell mechanism. Replacement of body cells seems to take place on a one-for-one basis, the cell which is being replaced apparently having some control on the new one. This type of replacement can be disturbed by an upset of the endocrine balance.

A seed is capable of building into organismal pattern the material it derives from the surrounding soil. In some soils it cannot grow, and the plant varies according to the nature of the soil which surrounds it. Certain soils produce marked differences in plants and their chemical constituents. Thus, animals fed on the same type of plants grown in different localities may also be effected.

In human beings and animals, the tissues show a tendency to return the structures to normal, as the replacement of lost bone structure in human beings and the replacement of dentine and enamel in the teeth of calcium-deficient animals.

Nature has a strong tendency toward restoring a lost balance to normal pattern. The cell chemistry and tissue replacement tend to restore the constitution of vegetable and animal tissue.

The deafness we have had under consideration is due to mechanical impairments of movable parts of the sound-conducting mechanism.

The cases reported show first normal mobility, second increasing immobility and third decreasing immobility of the moving parts of this mechanism.

There are biologic as well as clinical reasons for attributing the loss of mobility to physical changes in tissues in the region of the oval window, and the same reasons for attributing the later loss of immobility to changes of a similar nature. Biologic studies have revealed the mechanism of these changes in the tissue. The clinical phenomena herein presented are explicable on this basis.

DISCUSSION

DR. EDMUND P. FOWLER, New York: Dr. Lewis is striving to apply scientific facts and fancies to the variations of biochemistry and tissue change, in the hope that by trial and error he will succeed in changing local and general conditions. He reports favorably on two cases in which the patients were treated. Before we can pass judgment on the effects of his treatment, we must know the exact amount of hearing and the rate, variations and degree of loss over the fifteen or twenty years during which the deafness is reported to have increased—the family and personal history and the general otologic findings. If, after studying these, we are convinced that these are cases of stapes ankylosis and that real improvement has occurred, two main causes for improvement suggest themselves: (1) the one suggested by Dr. Lewis, that is, recession of lesions causing obstructive deafness, and (2) the improvement in general health and cessation of tinnitus. As far as is known, the progress of otosclerosis may be arrested but we do not know that there has ever been a recession or that the condition has disappeared.

DR. ARTHUR W. PROETZ, St. Louis (by invitation): Bilateral diseases occurring simultaneously are, as Dr. Lewis suggests, too common to be accidental.

While I follow Dr. Lewis' hypothesis that the bilateral incidence of fixation deafness suggests a predisposition to local tissue changes, in that systemic factors aggravate or at least permit rigidity in the conduction apparatus, I am not ready to accept the idea that improvement in the hearing is the result of an improvement in the local inflammation. It seems more likely that the tissue here follows a sequence such as occurs in inflammatory reactions, namely, congestion, hypertrophy, hyperplasia and finally atrophy, and that in the atrophic stage there may be less mechanical interference than in the hyperplastic stage, with consequent improvement in hearing.

DR. THOMAS J. HARRIS, New York: This contribution by Dr. Lewis is of great importance. It should receive our support and confirmation. I hope Dr. Lewis will make his studies available to our members, and especially that it will be put in the hands of our Committee on Progressive Deafness. I think that it deserves a place in our program next year for a further consideration of the subject.

DR. RALPH FENTON, Portland, Ore.: The extremely interesting speculations of Dr. Lewis regarding cases of fixation deafness which have shown definite improvement after metabolic changes were undertaken suggest that similar experimental treatment should be undertaken by other investigators, to whom, I feel sure, he is willing to give full details of his methods.

Undoubtedly, most of the hearing loss in these cases was due to an abnormal disposition of the connective tissue, perhaps for reasons inherent to the patient's own cellular set-up. Whether or not such cases come within the classic definition of otosclerosis and whether connective tissue which has become fixed into bone or scar tissue can be altered in reverse, we must applaud the fearless imagination with which Dr. Lewis has sought out methods of applying biochemical stimuli to such persons. Should it be possible to arrest the resistless progress of faulty calcification by the use of glandular products and appropriate diet, many types of research now going on will have to be scrapped. No doubt the essayist has it in mind to check accurately with a long series of similar cases, keeping accurate metabolic records along with several years' accumulation of audiograms. Only by such long-continued effort will the proof of his rather astonishing conclusions be made finally of record.

DR. EUGENE R. LEWIS, Los Angeles: I can assure Dr. Fowler that these patients have been subjected to the most searching scrutiny; they showed the usual signs of fixation of the stapes. I am unable to prove fixation except by functional test methods. Later we may get proof at autopsy. Dr. Fowler referred to its taking years to change a basal metabolic picture. I have seen a basal metabolic rate of minus 28 change to plus 18 in six or eight months.

Dr. Fenton mentioned otosclerosis. That term does not appear in my paper. In my opinion, the second case was definitely otosclerotic. Both patients showed deafness of the fixation type; that avoids discussing the exact nature of the cause. During the course of treatment, changes were noted in many general findings: blood pictures, body weights and the like. The second patient showed no change in hearing or tinnitus for eight months. She then began experiencing sudden cessation of tinnitus, during which "it sounded as if a window had been suddenly opened." Other patients have reported similar sensations. Dr. Harris mentioned the putting of this method into the hands of the Committee on Progressive Deafness. This paper is an attempt to do just that. The approach to the problem seems of prime importance, whether in a "localistic" or a "generalistic" frame of mind. A patient presents the effect of all factors whether hereditary or acquired, self-contained or enviromental, the exigencies of living and all others. The *status praesens* of the patient represents the product of all these factors. Inquiries must be made into the family and personal histories, past and present conditions, nutrition, exercise and domestic, psychic, emotional, business and environmental factors—anything tangible that can be pressed into service.

Book Reviews

Hirn und Ohr. By Karl Grahe, ä. o. Professor für Oto-Rhino-Laryngologie, Oberarzt der Universitäts-Ohren-Hals-Nasenklinik zu Frankfurt a. M. Price, 9.60 marks. Pp. 108, with 60 illustrations. Leipzig: Georg Thieme, 1932.

In this monograph the author undertakes to present in an introductory statement the anatomy and physiology of the peripheral labyrinth, the internal auditory meatus and the central pathways of the cochlear and vestibular nerves. This part is followed by a chapter on examination, including the voice hearing tests, the examination for tones and the equilibrium, with a discussion of the functional disturbances of the peripheral mechanism of hearing in diseases of the ears: first, the disturbances of the sound conduction mechanism, and second, the peripheral diseases of the labyrinth. The fourth chapter discusses the hearing and equilibrium disturbances of central origin, the central diseases, involvement of the eighth nerve, the pons, the medulla, the middle brain, the temporal lobes, the cerebellum, the frontal lobes and the general processes involving the brain, for example, intracranial pressure, vasomotor disturbances, meningeal inflammations, traumatism and other lesions of the brain. This brief presentation will be useful to the otologist as well as to the neurologist, the internist and the ophthalmologist.

Directory of Otolaryngologic Societies *

INTERNATIONAL

SECOND INTERNATIONAL OTOLARYNGOLOGICAL CONGRESS

President: Prof. Dr. A. G. Tapia, Hermosilla, 2, Mod, Madrid, Spain.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde, Heereweg 143, Groningen, Holland.
Place: Madrid, Spain. Time: September 27-30, 1932.

FOREIGN

NEW ZEALAND BRITISH MEDICAL ASSOCIATION, EAR, NOSE AND THROAT SECTION

Chairman: Dr. James Hardie Neil, Auckland.
Secretary: Dr. W. Gillies Borrie, Auckland.

ROYAL SOCIETY OF MEDICINE, SECTION OF LARYNGOLOGY

President: Dr. Walter G. Howarth, 21 Devonshire Place, W., London.
Secretary: Dr. A. Lowndes Yates, 33 Weymouth St., W. 1, London.

ROYAL SOCIETY OF MEDICINE, SECTION OF OTOLOGY

President: Dr. Norman Patterson, 82 Portland Pl., W. 1, London.
Secretary: Dr. T. B. Jobson, Netherwood, Epsom Road, Guildford.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Milwaukee. Time: June 12-16, 1933.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. S. Hanford McKee. 1528 Crescent St., Montreal, Canada.
President-Elect: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.

AMERICAN BRONCHOSCOPIC SOCIETY

President: Dr. Richmond McKinney, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Edwin McGinnis, 104 S. Michigan Ave., Chicago.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. George Fetterolf, 2010 Spruce St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Washington, D. C. Time: May 2-4, 1933.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. R. L. Loughran, 33 E. Sixty-Third St., New York.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. George L. Tobey, Jr., 270 Commonwealth Ave., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.
Place: Washington, D. C. Time: May 2-3, 1933.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

New England Otological and Laryngological Society

President: Dr. Henry A. Barnes, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. Seventh St., Los Angeles.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. John H. Harter, 1215 Fourth Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. R. B. Smith, Alma, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main Ave., Sioux Falls, S. D.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Sioux City, Iowa. Time: November, 1932.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Birmingham, Ala. Time: November, 1932.

Southwestern Michigan Triological Society

President: Dr. W. E. McGarvey, 802 Jackson City Bank Bldg., Jackson.
Secretary: Don C. Lyon, 1405 Union and Peoples National Bank Bldg., Jackson.
Time: Third Thursday of alternate months.

STATE

Colorado Oto-Laryngological Society

President: Dr. Harry L. Whitaker, 1612 Fremont St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. M. H. Gill, 36 Pearl St., Hartford.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Gillum, 221 S. Sixth St., Terre Haute.
Secretary: Dr. J. Kent Leasure, 611 Hume-Mansur Bldg., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: W. W. Pearson, 410, 6th Ave., Des Moines.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Harold J. Rothschild, 239 Lowry Bldg., St. Paul.
Secretary-Treasurer: Dr. W. E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month, October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. W. J. Marshall, 212 N. Higgins Ave., Missoula.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Time: Semi-annually.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. M. Hendershott, 193 Eleventh St., Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. McWhorter, 106 E. Earle St., Anderson.
Secretary-Treasurer: Dr. G. M. Truluck, Orangeburg.
Time: April and December.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas.
Secretary: Dr. A. F. Clark, 1034 Nix Bldg., San Antonio.
Place: Fort Worth. Time: Dec. 2-3, 1932.

Utah Ophthalmological Society

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

Virginia Society of Otolaryngology and Ophthalmology

President: Dr. Frank P. Smart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. Henry Grant Preston, Professional Bldg., Harrisonburg.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. L. C. Rouglin, 127 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore City Medical Society, Section on Otology and Laryngology

President: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis Diener, 2449 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

Buffalo Oto-Laryngological Society

President: Dr. John F. Healey, 925 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Herrmann E. Bozer, 468 Delaware Ave., Buffalo.
Place: Buffalo Club. Time: Second Tuesday of each month from September to May, inclusive.

Chicago Laryngological and Otological Society

President: Dr. Charles Younger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Cincinnati Oto-Laryngological Society

President: Dr. Henry M. Goodyear, The Doctors' Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

COLUMBUS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. F. Howland, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa
Time: Third Monday of each month, September to May, inclusive.

DETROIT OTO-LARYNGOLOGICAL SOCIETY

President: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. J. M. Sutherland, 8-257 General Motors Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Carl F. Snapp, Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: 6:30 p. m., third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. L. W. Raney, 609 Fannin St., Houston, Texas.
Secretary: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 314 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Mary Louise Cafe. Time: 6:30 p. m., fourth Monday of each month, May to November, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. Edward Leo Berger, 903 Park Pl., Brooklyn.
Secretary-Treasurer: Dr. Oscar Rodin, 893 Eastern Parkway, Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. T. F. McCormick, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: Dr. H. C. Smith, Medical Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month, October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Otolaryngology

Chairman: Dr. Charles Imperatori, 108 E. 38th St., New York.
Secretary: Dr. David H. Jones, 140 E. 54th St., New York.
Place: 103d St. and 5th Ave. Time: 8:30 p. m., third Wednesday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. L. G. Howard, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: Dinner, 6 p. m. Program, 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Louis H. Clerf, 128 S. Tenth St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. Roy C. Cooper, 510 S. Aiken Ave., Pittsburgh.
Secretary-Treasurer: Dr. Alexander R. Hampsey, 806 May Bldg., Pittsburgh.

Richmond Ophthalmological and Oto-laryngological Society

President: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis.
Secretary: Dr. B. J. McMahon, 813 Missouri Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Charles J. Boehs, 1126 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. L. Scales, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. David Hartin, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. W. W. Henderson, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 109 S. Warren St., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy W. Dunlap, 108 W. 6th St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. 6th St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.